

# Journal

## OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

*AVMA Convention—Cleveland, August 19-22, 1957*

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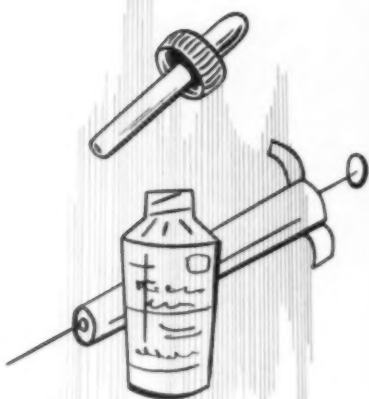
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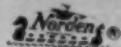
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**COMMITTEE ON JOURNAL:** T. Lloyd Jones, *Chairman*; Brigadier General Wayne O. Kester, W. W. Armistead (Board of Governors).

\$15.00 per Annum

Single Copies, \$ .75 Prepaid in U.S.

Canada and Pan American Countries, \$15.50; Foreign, \$16.50

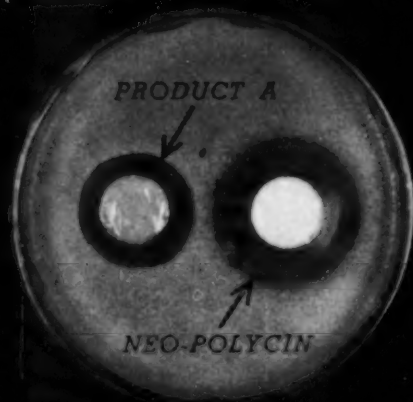
Published semimonthly at 600 S. Michigan Ave., Chicago 5, Ill., by the American Veterinary Medical Association. Second class mail privileges authorized at Chicago, Ill., January, 1956. Additional entry at Mendota, Ill.

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## *Tips on Working with Other Groups*

### Working with Health Agencies

D. R. MACKEY, D.V.M., Practitioner,  
Greeley, Colorado

Presented at the AVMA Public Relations  
Conference, San Antonio, Oct. 14, 1956

In the area in which I practice, Weld County, Colorado, we have participated in a program with the health agencies, which has been good public relations for us. I think such a program might benefit some of you.

First of all, you must have the public health agencies to work with. We have been fortunate in my locality, in having three separate units. First, we have a state public health unit and, in Greeley, where I reside, we have a county public health unit which is correlated with the state. Also in Greeley, we have a federal public health unit, a virus laboratory specifically, doing work on encephalitis. There is no other place where members are more willing to participate in our local associations than in the instance which I am going to mention. Also, it is a good opportunity to point out to nonmembers the advantages of membership in the associations.

### Cooperating with Public Health Agencies

Several years ago, they had a rabies outbreak in Denver, and the public health authorities became quite concerned about it. We had not had a case of rabies in Weld County for 50 years and, all of a sudden, two cases occurred.

The public health people came to the Weld County Veterinary Medical Association and wanted to know what we could do about it. They offered us a program. We discussed and "cussed" it. Some of the older members got very perturbed about working with the public health people, thinking that "if you give them an inch, they will take a mile," and "they won't turn us loose; they will take the thing over." But we finally convinced them that we ought to try it.

The public health people put on a public relations campaign to get pets vaccinated. They set up inoculation stations. They furnished the syringes; they furnished the needles; they furnished the sterilizers, the disinfectants, the clerical help; and they furnished someone to hold the dogs. As veterinarians, we furnished the vaccine and did the vaccinations for a very nominal fee, which probably to most of you would sound as though we were "off our rocker"—as it

did some of our members. But in a week's time we had 85 per cent of all the dogs in Weld County vaccinated. To this day we have not had another case of rabies.

That one experience brought our associations closer together. It also made the members realize the value of organized veterinary medicine, because we did work together.

Now that the epizootic of rabies in that area is past, the problem has been turned back to the practitioners and the vaccination is done in their offices. Many questions arise as to why it costs more to do it in the office than it did at the fire station.

There, again, a lot of veterinarians said, "Well, I hate to take time to explain that to every guy who comes in with his dog to be vaccinated."

That is where we are making a mistake. This is of public relations value. Let us not get tired of talking. Inform your people how much it costs to open the doors each morning. Inform them how much it costs to give a dog an inoculation.

Sometime back, the manager of our local hospital gave a talk on what it costs to operate a hospital, and I was very much surprised to find, from the statistics he gave, how much it costs to give a person a hypodermic injection. Discounting the value of what is in the syringe, it costs 35 cents—for breakage of syringe, breakage of needle, sterilization, the cost of the help to go from one place to another to give the injection, et cetera.

I believe many of us do not realize how much it costs us to open our doors each day.

In case of an emergency, when diseases do exist, we should forget about making a "fast buck" for ourselves and go out and conquer the disease, at least stop the spread of it and bring it under control, and do it as a public relations effort.

In the Weld County program, those who wanted to participate did so. The county associations furnished the vaccines. The veterinarian was paid according to the time he put in—not according to the number of dogs he vaccinated. The number of hours he worked was divided by the amount of

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#### Cooperating with the Livestock Boards

In addition to the public health agencies, we have a livestock group. There is a government range about 50 miles from Greeley, in which there are no veterinarians; yet, on that range, there is a vaccination program for cattle. All cattle put on that range must have been officially vaccinated as calves, or pass the blood test.

Again, the livestock boards of that area came to the veterinarians and said, "What will you do this for? If we organize and guarantee so many calves a day, how much will it cost?"

We gave them a figure. They guaranteed us 200 calves a day. The Weld County Veterinary Medical Association bought the vaccine. The veterinarians who wished to do the work took turns vaccinating those calves. In a week's time, we had all the calves ready to go on to the government range. We are going into the fourth year with that program.

The basis for paying those veterinarians for that work was the same as for the rabies program, that is, according to the time they gave to the program—not the number of animals they vaccinated. This is an especially good thing for the new man coming into the area, who is not too busy. It gives him a chance to meet livestock people and their officers, and to demonstrate his ability.

Too many times, when we face an outbreak, we as veterinarians want to capitalize on it, or at least make a little bit. But, really, fellows, if an emergency exists, we have a tool in the public health agencies that can put on an advertising campaign, get ordinances and laws passed to control these diseases. If we will meet our responsibilities in an emergency and not worry too much about how much are we going to make, but do it in the name of public relations, we can show the public what we as veterinarians can do.

#### Dog Shows

R. C. VIERHELLER, D.V.M., Practitioner,  
Whittier, California

"Can he walk? Then he must be all right." That is what one of the veterinarians said at a dog show to a lady who had a little dog under her arm. She went on her way with

the dog's identification card duly stamped—the veterinarian's certificate of good health.

This is the sort of burlesque on the science and name of veterinary medicine that is making a good many veterinarians decide they do not want dog show assignments.

In southern California, we have asked ourselves these questions:

1) Is the current employment of the show veterinarian one which reflects credit on the veterinary profession?

2) Is the best use of the show veterinarian being made?

3) Is it the best way we can manage this situation for the show veterinarian? In every case we have come up with a "no" answer.

#### The Time Provided Is Inadequate

The problem I refer to is the cursory, inadequate examination of dogs made as they enter the show. So little time is provided for each dog that all the veterinarian can do is give the dog a casual glance, stamp the identification card, and call that an examination.

In southern California, we set up a committee to study this problem in order to see how much time it would actually take. We found that we can not do an examination that would justify our certification in less than about six minutes, or 10 dogs per hour.

With dog show registrations today running from several hundred up to 2,000 or more dogs, the impossibility of this job becomes fairly obvious. The Long Beach show, for example, has something like 2,500 registrants. If we used our entire membership of 300 to examine these dogs in the couple of hours that are provided before the dogs are benched, we could not give them all an adequate examination.

#### Ethics

The question has arisen: "Is this action we are submitting to now ethical?" I believe it is a clear violation of ethics. Paragraph 27 of the AVMA Principles of Veterinary Medical Ethics has this to say: "The false certification of health on official documents shall be punishable by summary dismissal from the membership." It states further that "Careless compliance with official regulations that the veterinarian is en-



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trusted to enforce shall be deemed a violation of professional honesty."

Then, how long are we going to submit to this unethical conduct?

The utter ignominy of our position was brought out clearly at a dog show recently when I was there, not as the show veterinarian but simply as an investigator to study this problem. One of the veterinarians examined a dog that was led in by a very small boy. He stamped the card. As the little boy started to leave, he turned around, tugged on the veterinarian's sleeve and said, "Doctor, what can you tell by looking in my dog's face?" Not very much, I am afraid.

#### Do Exhibitors Show Sick Dogs?

Presumably, no exhibitor cares to risk the health of his dog by deliberately exposing it to the rigors of a dog show, when it is already sick. Certainly, if a dog is so sick that the veterinarian can spot that illness just by glancing at it as it passes by, it would appear sick to its owner and also to everyone else nearby.

But if we are going to have to conclude that some of the exhibitors will show the dogs anyway, in order to protect their entrance fee, then the time to examine those dogs is not at the show, after the dog is already exposed to many other dogs, but before the show.

The typical exhibitor at a dog show comes loaded with folding chairs, dogs, thermos bottle, lunch basket, combs, brushes, chalk, and all the other paraphernalia of his hobby. Instead of going first to the show veterinarian, if it is a bench show, he goes to the place where he is going to bench his dog and unloads his belongings. In the meantime, every dog in the vicinity has been exposed to his dog. About that time he will facetiously remark to his neighbor, "Well, I guess it is about time to walk my dog in front of the vet!" This is much too late to be of any benefit, if the dog is sick.

The professional handlers present a special problem. There is a serious question whether they are always able to present their charges to the show veterinarian. Professional handlers invariably arrive at the dog show with a station wagon full of dogs. They are under tremendous pressure to have these dogs in and out of the ring on a certain time schedule. The veterinary examination is a burden that he is either often unable to submit to, or he does not care to submit to it.

Many of these dogs come into the ring without the veterinarian's stamp on the identification card. Unless the ring stewards are particularly vigilant, these dogs are shown without examination. This, to me, would appear to be *prima facie* evidence that the examination rule is both unenforceable and useless.

#### What About Examinations?

Should the dogs be examined? I do not really know the answer to that. But if the veterinarians, the American Veterinary Medical Association, the American Kennel Club, and the exhibitors themselves believe the dogs should be examined, then they should be examined before they come to the show site.

A way of doing this would be to have them bring a certificate of good health from their own veterinarian, a preshow examination.

In discussing this method, we thought of the cost. The exhibitors are going to be up in arms if they have to pay for something additional. I think it would be very wise to have veterinarians give preshow examinations without charge for their regular clients, but not just for anybody. There is some precedent for doing this. I do not know about all areas, but in ours, we do examine dogs, without charge and as a good will gesture, which are taken from pounds and shelters. I believe we can afford to do that for dogs which are to be exhibited at shows.

Another question is whether remuneration for the veterinarian is part of this problem. I do not think so. In many cases, veterinarians are paid at dog shows now. I understand some local associations, and other groups, have demanded a fee for the veterinarians at dog shows and have received it, but that does not change the problem. It is still the same degrading situation. On top of this, the exhibitor knows that a chunk of his entry fee is being paid to the veterinarian for something to which he can not attach much value.

#### Should Veterinarians Be at Dog Shows?

Definitely, veterinarians should be there. It would be a serious reflection on our interest in animal matters and would be bad for our public relations if we should refuse to be there. We have a place at dog shows, but we should be there in a way that re-

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How are we going to accomplish this? I believe the method that we can use is to set up a field hospital, or well-equipped first aid station, at dog shows. Dogs do cut their feet on broken bottles and get into fights; in warm weather, there are certain breeds that are prone to heat prostration. Occasionally, a dog becomes sick suddenly and a veterinarian's decision is necessary as to whether or not it should be removed promptly. So, we have a place at dog shows.

The judges, too, like to bring dogs for a count of certain parts of the anatomy—for the veterinarians to decide whether they are intact or not.

We have a place at shows but we should be prepared to handle our responsibility adequately.

In conclusion, the organization that is best suited to set up a program of this kind for its members is the American Veterinary Medical Association. This program should include these four points:

1) Elimination of the cursory, unprofessional, and unethical examination now being given dogs at dog shows.

2) Provision for a field hospital adequately equipped to take care of the dogs at shows licensed by the American Kennel Club. This could also be used as a point to disseminate public relations material, such as the activities and purposes of our societies, and helpful information, such as dog pamphlets on care, feeding, and immunization.

3) Substitution of a preshow examination for the examination at the show. This is only to be applicable if we feel that an examination is really necessary, and it should have a time limit, so that the examination will not be too far in advance of the show. Possibly three days would provide adequate time for the owner to see his veterinarian for an examination. The time should be short enough so that illness of the animal in the intervening time would be unlikely.

4) Cooperation with, and aid to, the American Kennel Club in rewriting its rules pertaining to the show veterinarian.

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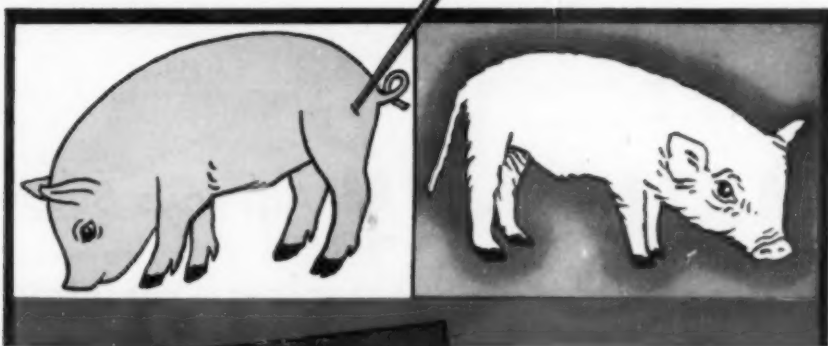
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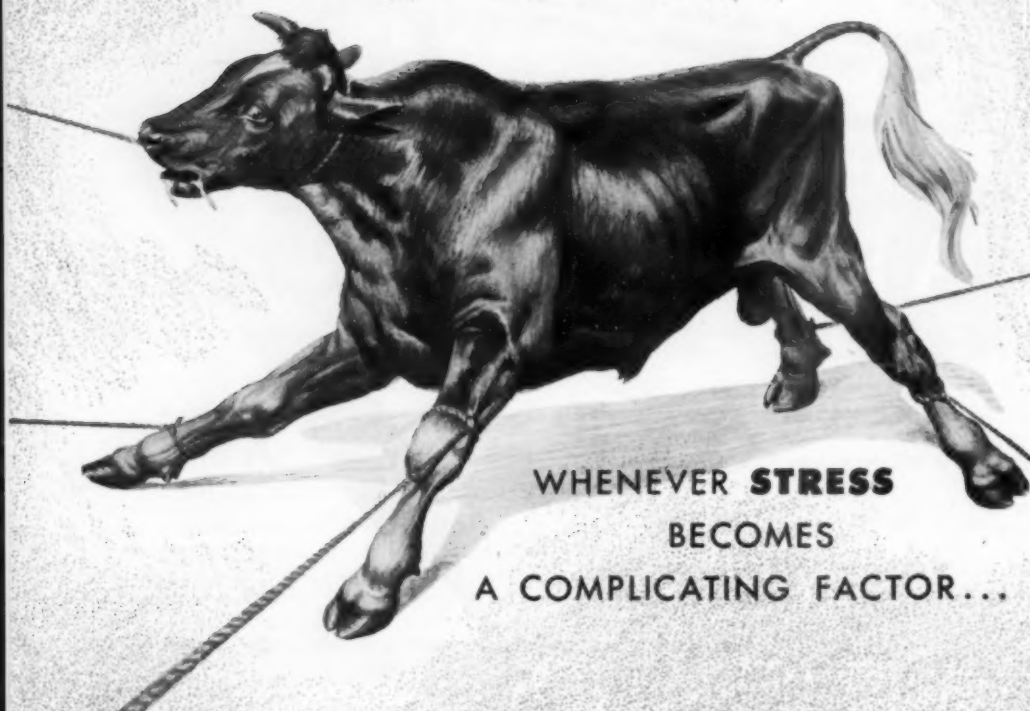
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M-49-56-5

Hobbs—Female—1 week of age

DIAGNOSIS: CALF SEPTICEMIA

HISTORY:  
Calf penned with other calves near a north door.  
Five of nine calves developed scours and septicemia.

SYMPTOMS:  
Temperature 100° F., pulse 100 and respiration 60.  
Animal unable to stand, mouth cold to touch.

TREATMENT:  
5 cc. of METICILLIN, 2 Gm. of streptomycin intramuscularly and 2 VARITON Compound boluses were administered

RESULTS:  
Calf up and offering to eat 16 hours later. One VARITON Compound bolus given twice a day for two days.

Case M-5-56-6

Canine—Mixed—Male—3 months of age

DIAGNOSIS: DISTEMPER

HISTORY:  
From animal shelter.

SYMPTOMS:  
Temperature 103.4° F., typical catarrhal distemper symptoms.

TREATMENT:  
1 cc. METICILLIN daily for 3 days. Supplemental therapy given the first two days included 15 cc. of antidiarrheal serum and 1 cc. of B-complex.

RESULTS:  
Excellent. Catarrhal discharge disappeared at end of the second day. Temperature dropped to normal within 24 hours after the first dose of METICILLIN and appetite returned. Animal dismissed on 4th day of hospitalization.

C-13-56-5

Jersey—Female—2 years of age

DIAGNOSIS: HEMORRHAGIC SEPTICEMIA

HISTORY:  
Animal purchased at stock sale 5 days before first examination. Treated with tetracycline intraperitoneally two days previously. Condition improved, but animal relapsed until her condition was more severe than in original state.

SYMPTOMS:  
Definite evidence of pneumonia. Temperature 105° F., increased respiration and pulse, animal not eating.

TREATMENT:  
One vial (10 cc.) of METICILLIN administered intramuscularly and 1½ gr. of tetracycline administered peritoneally.

RESULTS:  
Animal was eating by the following day and made an uneventful recovery.

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\*Link, R. P.; Newton, D. I., and Huber, W. G.: The Use of Prednisolone in Bovine Ketosis, paper presented at the 93rd Annual Meeting, A.V.M.A., San Antonio, Texas, Oct. 15-18, 1956.

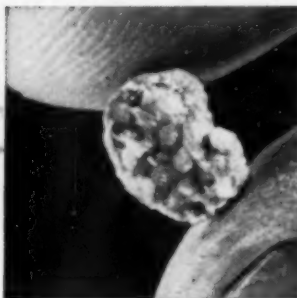


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\*Jones, S. V.; Belloff, G. B., and Roberts, H. D. B.; Vet. Med. 51:413 (Sept.) 1956.


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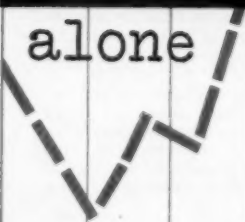
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Welch, H.: F. D. C. Reports 18:8, 1956.

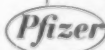
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One clinical study of 328 cases of milk fever treated showed that Parterol I.M. and calcium I.V. were effective in 98% of the cases.<sup>1</sup>

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To prevent milk fever by regulating the calcium blood and tissue level at the critical calcium-utilization period at parturition. Parterol should be given 24 hours prior to parturition in a dosage of 10 mg.

Supplied: In 30 cc. sterile vials, each cc. containing 2.5 mg. dihydrotachysterol in oil.

1. Harris, J.R. and Clarkson, T.B., Prevention of Relapses in Milk Fever, Vet. Medicine, 12:696 (Dec. 1955)

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**\*Patent applied for**

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<i>Thoroughbred Gelding</i> <sup>*</sup> amputate scirrhus cord	bleeding despite ligatures—10 cc. I.V., 10 cc. I.M.	"Seeping hemorrhage was arrested in 10 to 15 minutes."
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<sup>\*</sup>N.B. A second thoroughbred under identical conditions but without KOAGAMIN: postoperative seeping hemorrhage—½ hour; oozing—an additional 2 hours.

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1. Rachman, M., and Frucht, T. R.: Vet. Med. 49:341, 1954.



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# JOURNAL

of the American Veterinary Medical Association

Established January, 1877

Chicago 5, Illinois

Vol. 130

MARCH 1, 1957

No. 5

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## Experiments and Observations Leading to Development of the California Mastitis Test\*

O. W. SCHALM, D.V.M., Ph.D., and D. O. NOORLANDER, B.S.

*Davis, California*

THERE HAS BEEN a need for a quick, reliable test for the detection of abnormal milk at the side of the cow. To be of value, the reaction should be instantaneous and sufficiently clear-cut to leave no doubt as to whether the milk is normal or abnormal. It was with this in mind that the studies reported herein were undertaken.

In searching for an accurate mastitis test for use in the dairy barn, it was natural that an attempt should have been made to adapt the Whiteside phenomenon<sup>1</sup> to this need. The modified Whiteside test<sup>2</sup> consists of using 1 drop of 4 per cent sodium hydroxide and 5 drops of cold milk, or 2 drops of NaOH and 5 drops of warm milk, on a glass plate and stirring vigorously for about 20 seconds. A positive test is recorded when the milk thickens and then separates into flakes or shreds and a semiopaque to clear whey.

To bring the Whiteside test closer to the cow, it was suggested that the test be conducted in a glass vial or tube.<sup>3</sup> The vials were prepared before going to the dairy by placing 2 ml. of 4 per cent sodium hydroxide containing cresol red for color contrast in each. Milk was drawn to the 12-ml. level using a single tube for each quarter, then the tubes were immediately inverted several times to mix their contents. A positive reaction was recorded when an immediate thickening developed or when shreds or flakes appeared on the exposed glass wall. This procedure was designated the "field Whiteside test"; in routine use, it was

found to correlate well with the modified Whiteside test. There were some samples which gave indications of probable positive reactions but the particulate material was indefinite or difficult to detect. A further disadvantage was that the milk, both normal and abnormal, tended to gel after a few minutes of contact with the sodium hydroxide.

### DEVELOPMENT AND USE OF MASTITIS TEST FORMULA 2

Attention was directed toward development of a test solution that would overcome the disadvantages of 4 per cent NaOH as used in the field Whiteside test. It was conjectured that a surface-active agent added to the sodium hydroxide might enhance the release of the abnormal materials in mastitic milk, thus causing the flakes and shreds to become more prominent. An anionic surface-active agent, such as an alkyl arylsulfonate, was found to improve materially the field Whiteside test. The dry form of detergent (0.5%) and NaOH (1.5%), to which cresol red was added to give color contrast, proved to be much more sensitive than 4 per cent NaOH for detection of milk of abnormal cell counts; also, the milk did not gel upon prolonged contact with the reagent. This was designated formula 2 to distinguish it from 4 per cent NaOH normally employed in the Whiteside test. When a test for mastitis is to be conducted on milk in a test tube, formula 2, added to the milk in the ratio of 1 to 1, is highly satisfactory.

Since the handling of much glassware in the dairy barn is inconvenient and expensive, a plastic paddle fitted with four cups or milk receptacles was devised. The

From the School of Veterinary Medicine, University of California, Davis.

\*The Regents of the University of California have filed patent application on the California mastitis test. This was done in order to limit distribution of the test through the veterinary profession.

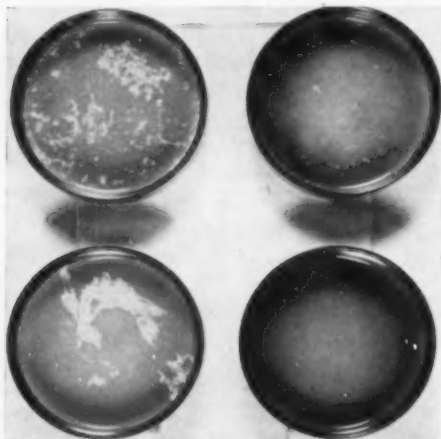


Fig. 1.—Reactions produced in milk with mastitis test solution formula 2. The reactions are scored as follows: lower right, negative; upper right, 1; upper left, 2; and lower left, 3. Appearance of positive reactions in the black cups of the paddle, as shown here, is similar to results obtained with the modified Whiteside test conducted on the surface of a glass plate.

first paddle consisted of a piece of clear plastic to serve as a handle and base to which four black bakelite chemical jar lids were affixed (fig. 1).

About 2 ml. of foremilk was drawn from each teat into the respective cup and formula 2 was added in equal volume, by estimation, by squirting it from a polyethylene wash bottle. Mixing was accomplished by a gentle circular motion of the paddle in the horizontal plane. With mastitic milk, there occurred an initial thickening, the degree of which was in direct proportion to the cell count of the milk. As the motion of the paddle was continued, the initial thickening disappeared and, at the same time, the opacity became reduced or vanished entirely and whitish flakes or shreds floated to the surface of the liquid. Since the initial thickening was momentary, except with milk of exceedingly high cell count, it was natural to tend to overlook its occurrence and attempt to score the reaction of positive milk on the basis of the amount of shredding or clumping of the particulate material which formed in the secondary phase of the reaction. In comparing the reactions so scored with actual cell counts in the milk, there was, in general, a good direct correlation. However, it became apparent that with some milk the

reaction scores were significantly less severe than the magnitude of the cell count justified. Disagreements between formula 2 reaction scores in the paddle and actual cell counts in the milk occurred more often with foremilk than with samples of mixed milk of the entire udder. This suggested that perhaps milk fat might be involved in the visible phase of the Whiteside phenomenon.

#### OBSERVATIONS ON THE NATURE OF THE WHITESIDE PHENOMENON

One gram of whitish particulate material released from mastitic milk by the addition of formula 2 in equal parts was assayed and found to consist of 43 per cent fat. This finding further suggested that the milk fat is involved in the formation of the shreds and clumps which occur in mastitic milk in the presence of dilute sodium hydroxide. Therefore, the following experiment was undertaken. Milk, strongly positive to the Whiteside test, was centrifuged at 1,800 r.p.m. for 20 minutes. The fat which rose to the surface was removed by suction. Without disturbing the sediment, the skimmed milk was poured off and tested for Whiteside reaction. A slight thickening occurred but this immediately disappeared, leaving a clear fluid devoid of any particles as commonly seen in the secondary phase of the Whiteside phenomenon. Next, skimmed milk and sediment were mixed and sediment alone was suspended in distilled water; when sodium hydroxide was added, as in the modified Whiteside test, both samples showed distinct thickening followed by disappearance of the viscosity and occurrence of a few very delicate, almost colorless strands. Finally, fat taken from centrifuged normal milk was used to replace fat removed from centrifuged mastitic milk; it was demonstrated that the fat from normal milk participated in the secondary phase of the Whiteside reaction in the same manner as the original fat of the mastitic milk.

The phenomenon whereby fat globules in mastitic milk are acted upon by sodium hydroxide to form strings or clusters was demonstrated under the low power of the microscope. A small drop of normal milk and another of mastitic milk were placed under separate cover slips. The 4 per cent NaOH was placed at one edge of each cover slip in a manner to permit it to be drawn under the cover slip by capillary action.

Under high power magnification, the leukocytes in the mastitic milk disintegrated upon contact with the NaOH. After the NaOH solution had moved across the milk film, the cover slips were moved in a circular fashion to agitate the mixture of milk and NaOH. In the mastitic milk, the fat globules rushed together, forming strings and isolated clumps (fig. 2); whereas in the normal milk, the globules remained dispersed.

#### THE CALIFORNIA MASTITIS TEST

From the observations recorded above, it appeared that a reagent was needed, for use with the paddle, that would not involve the milk fat as part of the visible positive reaction with mastitic milk. Attention was turned to the anionic surface-active agent which had been employed in mastitis test formula 2. It was found that this reagent in a 3 to 5 per cent concentration reacted with milk of high cell count in a manner that could be graded satisfactorily. The reaction varied in degree from slight precipitation of amorphous material, that tended to disappear again, to immediate development of a viscoid gel.

After many trials with a number of surface-active agents, it was concluded that this particular reaction might be anticipated when milk of high cell count is brought in contact with the sodium or potassium salts of any of the following in proper concentration: long chain fatty acids, alkyl sulfates, alkyl sulfonates, alkyl arylsulfates, or alkyl arylsulfonates.

By employing an anionic surface-active agent of pH near the neutral point, an in-

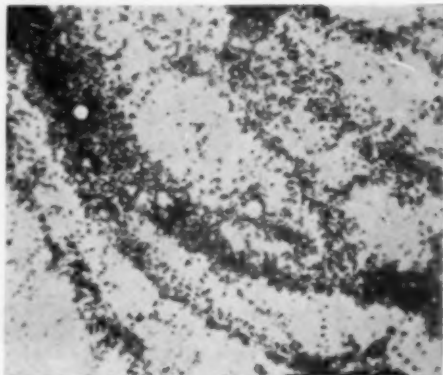


Fig. 2—Fat globules forming shreds and clumps, following addition of 4 per cent sodium hydroxide to mastitic milk under a cover slip on a glass slide. Low power magnification of microscope.  $\times 100$ .

dicator, such as bromcresol purple, may be added to reveal abnormal alkalinity or acidity of the milk and, at the same time, a contrasting color is obtained. Bromcresol purple (dibromo-o-cresolsulfonephthalein) in final concentration of 1:10,000 provides a good contrasting color with milk against a white background.

A white plastic paddle is required for use with the new reagent which was designated C.M.T. for California mastitis test. The base of the paddle is  $6\frac{1}{4}$  inches square and  $\frac{1}{8}$  inch thick. Each milk receptacle or cup has an outside diameter of 3 inches and is  $\frac{3}{4}$  inches deep. The milk receptacles are separated from each other by a  $\frac{1}{4}$ -inch space at their closest points. This space is necessary in order to permit drain-

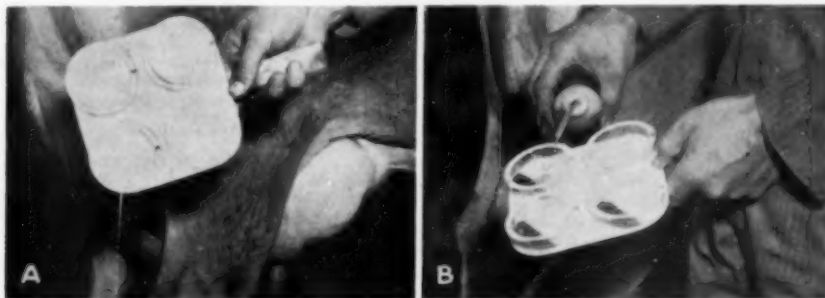


Fig. 3—The California mastitis test is conducted on milk from individual quarters drawn into the respective cups of a white plastic paddle. After the excess milk is drawn off (A), the reagent is added in equal volume to the milk by squirting from a polyethylene wash bottle (B). A precipitate or gel forms in milk of high cell count as mixing of milk and reagent is brought about by gentle circular motion of the paddle.

ing excess milk from one or more of the cups when too much milk has been drawn from the teat into the respective cup.

**Procedure.**—Foremilk is normally used although strippings may be employed. About 2 to 3 ml. of milk is drawn directly from the teat into the respective cup. Too much milk interferes with accurate scoring of the reaction. In the event excess milk has been drawn, the ideal quantity can be obtained easily by tipping the paddle toward the vertical, thus permitting the excess milk to drain away (fig. 3, B). The reagent is squirted into the milk from a polyethylene wash bottle (fig. 3, A). It is important to add, by estimation, a quantity of reagent at least equal in volume to the milk in the cup. The mixture of reagent and milk is made to swirl by a gentle circular motion of the paddle. Positive reactions develop immediately and the milk is scored during the movement of the paddle. The paddle is rinsed in cold water and is ready for use again without drying; a trace of moisture will not interfere with the test.

At the side of the cow, the test should be applied as a mastitis test only during the period of active lactation. It is best not to apply the test until the third day after calving and not after drying-off has been started. Both colostrum and the secretion of the drying-off gland are high in cell content and, therefore, positive C.M.T. reactions may be obtained with such secretions.

While this test was developed for use at the side of the cow, it may be used in the laboratory on samples of bucket milk, that is, the mixed milk of the entire udder, or on bulk milk as delivered to the creamery. Bacterial growth in milk will destroy the factor or factors that support a positive reaction with C.M.T. Therefore, when bucket or bulk milk samples are tested, they should be refrigerated to control bacterial growth and the test should be conducted within 24 to 36 hours for best results. When large numbers of bucket or bulk milk samples were tested routinely, it was found convenient to use a ten-cup plastic unit and to accomplish mixing mechanically (fig. 4) by means of a device made from a record player.

**Explanation of Positive Reactions.**—Two reactions are to be noted—formation of a precipitate or gel and the development of contrasting shades of purple between the milk from the four quarters. The reagent

ruptures cells, releasing the cellular protein; the corpuscular proteins are unfolded as a result of a breaking of bonds and these unfolded protein molecules unite with the reagent causing it to precipitate or gel. The reaction proceeds best in a neutral or slightly alkaline pH. This is the pH of most mastitic milk. The pH of normal milk is in the range of 6.4 to 6.8. If the reagent *per se* is neutral or slightly alkaline, normal milk will act as a buffer and bring the pH of the mixture toward that of the milk and the color imparted by the bromocresol purple will be blue-gray. On the other hand, if the milk is above pH 6.8, which is often the case in chronic mastitis, the bromocresol purple will impart a purple color to the mixture. Alkaline milk always indicates depression of milk secretion.

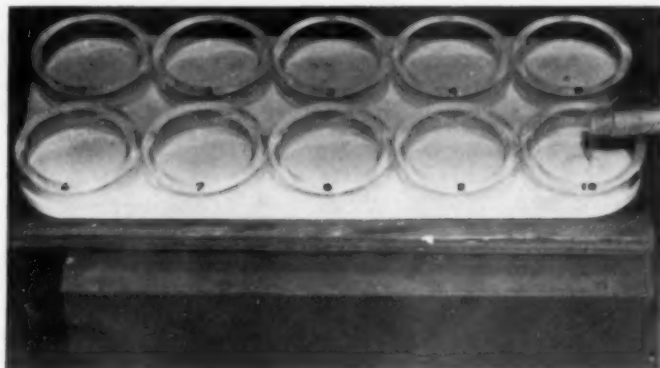
#### Scoring C.M.T. Reactions

Negative (—)	Mixture remains liquid with no evidence of formation of a precipitate.
Trace (T)	A slight precipitate which tends to disappear with continued movement of the paddle.
Weak positive (1)	A distinct precipitate but no tendency toward gel formation.
Distinct positive (2)	The mixture thickens immediately with some suggestion of gel formation. As the mixture is caused to swirl, it tends to move in toward the center, leaving the bottom of the outer edge of the cup exposed. When the motion is stopped, the mixture levels out again covering the bottom of the cup.
Strong positive (3)	A distinct gel forms which tends to adhere to the bottom of the paddle and, during swirling, a distinct central peak forms.
Alkaline milk (+)	The plus sign should be added to the score when the mixture is distinctly purple as indicated by a contrasting deep purple color.
Acid milk (y)	Bromocresol purple is yellow at pH 5.2. Infrequently, milk is acid when drawn from the teat. Such an occurrence should be reported and may be indicated by the letter "y."

#### RESULTS AND DISCUSSION

California mastitis test results, with mean total cell counts per milliliter of milk, on 573 foremilk, 136 stripping milk, 234 bucket, and 154 bulk samples are compared (table 1). Also, the mean percentage of occurrence of the polymorphonuclear (PMN) leukocyte (pus cell) is shown. In normal foremilk, that is, milk negative to the test, the mean total count did not

Fig. 4—A ten-cup plastic unit and mechanical device for mixing the milk and reagent by gentle circular motion. This means for conducting the California mastitis test is used when large numbers of bucket milk or bulk milk samples are tested routinely in the laboratory or at the creamery.



exceed 100,000 cells per milliliter of milk and the percentage of PMN leukocytes was less than 25. With C.M.T. negative stripplings, the mean total cell count per milliliter of milk was twice that of negative foremilk but the PMN leukocytes remained less than 25 per cent of the total cell count. On the other hand, negative bucket milk showed mean PMN leukocyte counts near 40 per cent. What this actually indicates is that with bucket milk a negative reaction does not necessarily mean that all quarters of the udder are normal. Partially atrophied quarters may not contribute a sufficient volume of secretion to the mixed milk to cause it to support a positive reaction,

although by microscopic cell count the increased occurrence of PMN leukocytes over normal would suggest that the milk was not from an entirely normal udder. Of the 154 samples of bulk milk, only eight were entirely negative. Due to age of the milk and condition of the cells in the milk, a differentiation of cell types was not attempted. The grading of positive reactions in bulk milk was modified in order to compensate for the effect of dilution, so that a score of 3 was actually equal in intensity of reaction of a strong 1 or weak 2 as encountered in foremilk.

The results as presented in table 1 show that as the reaction becomes more

TABLE 1—Comparison of C.M.T. Score with Mean Total Cell Count Per Milliliter of Milk and Percentage of Polymorphonuclear (PMN) Cells

Type, No. samples, and source	Type of data	Mean cell count/ml. compared with C.M.T. score				
		Negative	Trace	1	2	3
Quarter foremilk, 481 samples, herd SSH	No. of samples	271	52	95	43	20
	Total cells/ml.	81,400	431,000	1,157,000	3,250,000	11,435,000
	Percentage PMN cells	23	31	48	61	77
Quarter foremilk, 92 samples, assembled herd	No. of samples	54	2	10	10	16
	Total cells/ml.	87,000	500,000	753,000	3,590,000	12,400,000
	Percentage PMN cells	23	20	66	43 (53)*	68
Quarter stripplings, 136 samples, herd SSH	No. of samples	32	37	43	13	11
	Total cells/ml.	169,000	428,000	1,121,000	4,310,000	9,580,000
	Percentage PMN cells	21	37	49	70	70
Bucket milk, 113 samples, herd S.Q.	No. of samples	61	19	21	9	3
	Total cells/ml.	129,000	310,000	690,000	1,970,000	8,500,000
	Percentage PMN cells	39	35	48	67	70
Bucket milk, 121 samples, herd UCF	No. of samples	56	19	28	18	20
	Total cells/ml.	152,000	207,000	1,050,000	2,720,000	8,290,000
	Percentage PMN cells	40	42	52	63	73
Bulk farm milk,† 154 samples, 61 different herds	No. of samples	8	23	51	47	25
	Total cells/ml.	250,000	550,000	720,000	1,130,000	1,480,000

\*Three of these quarters showed advanced atrophy and watery secretion; the mean total cell count was 2,570,000 with only 21 per cent PMN. The secretory tissue had been destroyed and the inflammation subsided. The mean cell count of the milk from seven remaining quarters was 4,030,000 with 53 per cent PMN cells.

†The grading of positive reactions in bulk milk was modified in order to compensate for the effect of dilution, aging, and temperature on the factor responsible for the positive reaction. A 3 score was equal in intensity of reaction to a strong 1 or weak 2 as encountered with quarter sample milk.

pronounced, both the total cell count and percentage of PMN leukocytes increase. Therefore, C.M.T. can be used as a rapid means for screening of milk for poor quality. It may be employed by the creamery on bulk milk for detection of herds having a high degree of udder irritation; it may be used on bucket samples to screen a dairy herd for cows producing milk of significantly high cell count; and on the individual quarters of the udder to detect the abnormal quarters.

Programs for the prevention and control of mastitis may be planned around the routine use of C.M.T. on all lactating cows. With larger commercial herds, it has been found convenient to instruct the herdsman in the use of the test. Through using it at weekly intervals and recording the results on a master sheet, it has been possible to detect mastitis in the incipient stage and to locate all quarters affected with chronic mastitis. In addition, the checking of the udder of the cow that goes off feed or the recently fresh cow in which milk production decreases, may call attention to the existence of acute mastitis much earlier than would otherwise be known.

The California mastitis test is a very sensitive indicator of the presence of inflammation in the udder. It can not, however, tell the cause of the inflammation. Udder irritation due to improper mechanical milking will result in positive reactions, as will also inflammation due to bacterial infection.

In herds showing a high incidence of positive glands, the first step should be to correct milking techniques. Glands that fail to improve should be sampled for bacteriological study and treated with specific therapy as indicated by the findings. Quarters that continue to produce an alkaline milk and a strong C.M.T. reaction, despite all efforts to benefit them, might best be dried-off. A long period of rest before the next lactation begins may return the gland to better health.

#### SUMMARY

There has been a need for a rapid, accurate test for mastitis for use on milk at the side of the cow. This paper reports the development of such a test which has been designated the California mastitis test (C.M.T.).

A white plastic paddle with four receptacles, into which the milk is drawn direct-

ly from the respective teat, is used. The test solution is added in an estimated equal quantity to the milk in each cup by squirting the reagent from a polyethylene wash bottle. The reactions occur and are graded, while the milk and reagent are mixed by a gentle circular motion of the paddle.

The cell count of the milk is reflected by the degree of precipitation or gel formation. The extent of depression of milk secretion is indicated by color reaction with the bromocresol purple in the formula, due to alkalinity.

In addition to its use with foremilk or strippings of individual glands, the test is applicable to bucket milk (mixed milk of an udder) for rapid screening of herds for mastitic cows, and to bulk milk as delivered to the creamery for the selection of herds having a high degree of udder irritation.

#### References

- <sup>1</sup>Whiteside, W. H.: Observations on a New Test for the Presence of Mastitis in Milk. *Canad. Pub. Health J.*, 30, (1939): 44.
- <sup>2</sup>Murphy, J. M., and Hanson, J. J.: A Modified Whiteside Test for the Detection of Chronic Bovine Mastitis. *Cornell Vet.*, 31, (1941): 47.
- <sup>3</sup>Schalm, O. W., Gray, D. M., and Noorlander, D. O.: Procedures for the Use of the Whiteside Test on Milk in the Laboratory or Barn. *North Am. Vet.*, 36, (1955): 1011.

#### Mastitis During the Dry Period

Secretion samples were examined weekly from 75 cows during their dry period. New infections were chiefly due to *Streptococcus agalactiae*. If there is a natural sealing of the teat orifice, it is of minor importance in preventing infection. The rate of new infection increased in ratio with the yield when milking was terminated. New infections were more common in cows that had been milked by hand.

When, after the last milking, the udders of alternate cows were washed with sodium hypochlorite solution (880 p.p.m.) and then dried, the teats immersed in a 5 per cent iodine tincture for 20 seconds, and the treatment repeated in 24 hours, the reduction in infection in the treated cows was highly significant.—*Vet. Bull.*, Nov., 1956.

Successful treatment of 18 cases of bovine mastitis, simply by anesthetizing the lumbar nerves to the udder, is reported from Poland.—*Vet. Bull.*, Dec., 1956.

## Interpretation of Production Graphs in Mastitis Control\*

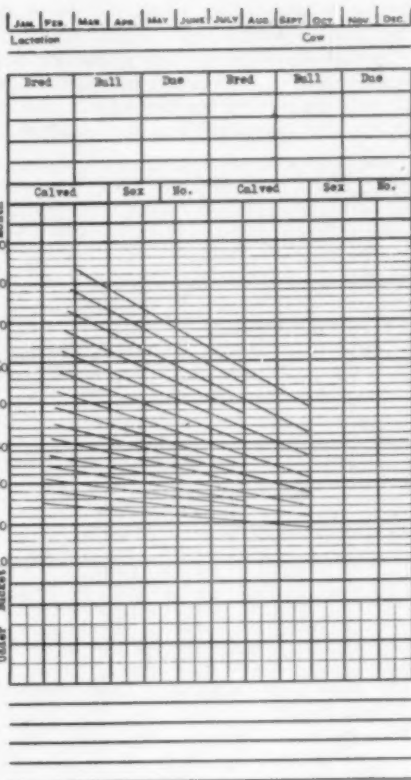
A graph of a cow's production is actually a picture of her performance. Such graphs, based upon the weight of the milk produced in a 24-hour period and obtained once each month, when combined with an adequate mastitis test, provide a basis for (1) evaluating the effect of mastitis on the quantity of milk secreted and (2) measuring the improvements in individual cows and the herd as a whole following the use of measures for the control and treatment of mastitis (graph 1).

Milk graphs of normal cows present patterns that are distinctly different from those now commonly found among cows in a majority of commercial dairy herds. Mastitis is so rampant in present-day herds that few cows show normal production patterns. The normal milk graph of the mature cow shows a sharp rise during the first month or two of lactation, followed by a gradual but uniform decline until the effect of advancing pregnancy (about the fifth month) causes the secretion to fall off more rapidly.

Mastitis severely restricts maximum development of the udder. Some cows never exceed their first lactation record, and most cows produce less milk by the third and fourth lactations than they did in the preceding ones.

There is a significant falling-off of milk secretion long before signs of clinical mastitis appear.

\*Excerpts from a leaflet, "Use and Interpretation of Production Graphs in Mastitis Control," published by the School of Veterinary Medicine, University of California, Davis. The complete leaflet is available at 5 cents per copy, and graph sheets at 2 cents each, from: Associated Students Store, University of California, Davis, Calif.

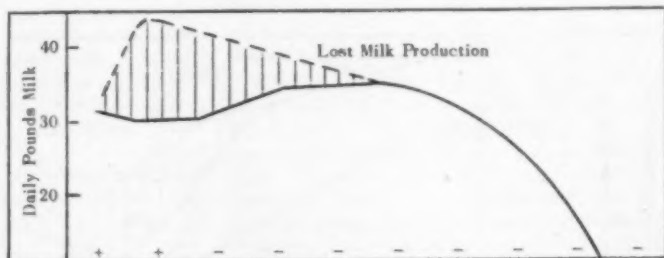


School of Veterinary Medicine, University of California, Davis

Graph 1—When a cow's production is recorded on such a graph, it constitutes a picture of her performance.

Marked decline in milk production in the absence of a reaction to the mastitis test in the individual quarters of the udder should cause the dairyman to evaluate his feeding program. If this is found to be adequate, an examination for the existence of other

Graph 2—A flat milk graph in the mature cow is abnormal.



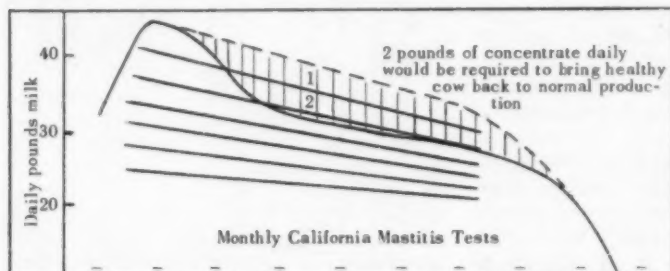
diseases should be made by the veterinarian.

*Graph 2, the Flat Milk Graph.*†—A flat milk graph in the mature cow is abnormal. A flat line, except for first-calf heifers, denotes that the cow has not reached her

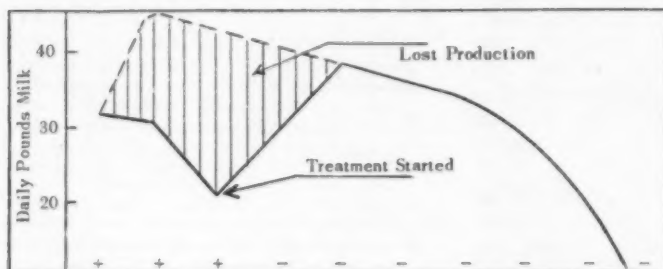
†We have used graphs 2, 4, 6, 7, and 8 from the original leaflet and have renumbered them 2, 3, 4, 5, and 6, respectively.

potential for production in early lactation. This is typical of cows that enter lactation with the udder severely affected by mastitis.

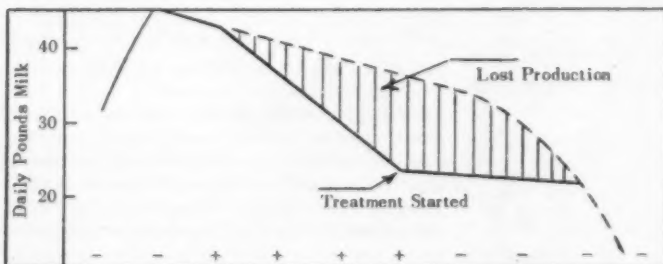
*Graph 3, Feeding to Support Maximum Production.*—The stimulus for milk secretion is so intense in early lactation that most cows in good flesh will reach their



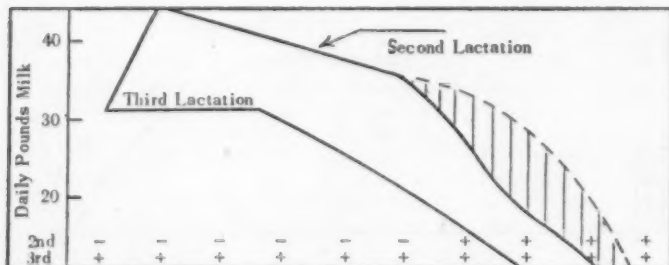
Graph 3—Feeding to support maximum production.



Graph 4—Milk graph typical of cows given early treatment for mastitis.



Graph 5—Milk graph typical of cows given treatments for mastitis beyond the midpoint of lactation.



Graph 6—This graph is typical of that for cows with chronic mastitis that have received no treatment.

maximum production at the expense of body tissue. As soon as the body reserves for milk production have been depleted, the volume of milk secreted will drop to the level that can be supported by the feed consumed. Thus, milk graphs of normal cows that are underfed will drop below the normal graph line. The diagonal lines on the graphing sheet are so spaced that they can be used to determine the approximate pounds of concentrate to feed daily to bring an underfed cow up to normal production. Therefore, for each diagonal line to which production has declined from the anticipated potential, an additional pound of concentrate should be fed daily to supply the feed requirement for maximum milk secretion.

*Graphs 4 to 6, Treatment of Mastitis.*—Proper and early treatment of mastitis is important to maintain milk production at near normal level. The veterinarian should make a bacteriological study of milk from the mastitic glands to determine the type or types of bacteria present, so that the most effective treatment can be selected. Graphs 4 and 5 depict types of milk graphs that may be anticipated in cows given treatment for mastitis. Graph 6 is typical of cows with chronic mastitis that are allowed to go untreated. Severe damage has resulted so that the volume of milk secreted in the third lactation is reduced and the milk is inferior in quality as well.—*D. O. Noorlander, B.S., and O. W. Schalm, D.V.M., Ph.D., Davis, Calif.*

Intravenous injection of 250 to 500 ml. of glucose solution (50%) daily for six days had no apparent effect on the production of milk or milk fat in normal cows.—*Guernsey Breeders' J., Sept. 15, 1956.*

### Cutaneous Anthrax in Man

In seven years, in Kenya, Africa, 502 persons were treated for cutaneous anthrax at one hospital; most of them were members of a nomadic, cattle-raising tribe. Most of the lesions were typical malignant pustules but a few were "itching sores" resembling scratched insect bites. One child with an itching lesion, who was given local treatment only, returned the next day with a massive local edema, a temperature of 104 F., and convulsions (not uncommon in affected children).

Of the 502 persons treated, only three died—all small children. Adults respond

well to procaine penicillin, 300,000 units daily until they become afebrile, but children require a heavier dosage (900,000 units daily).—*Brit. Med. J., Dec., 1956.*

### Short Reports on Zoonoses

Infectious encephalitis in man was about 50 per cent more prevalent in 1956 than in 1955. Eastern equine encephalomyelitis in human beings, principally in children, was most common in Massachusetts and Maryland. The virus was isolated from the brain tissue of persons who died from the infection. The disease was confirmed in horses in Massachusetts, Maryland, New Jersey, Delaware, and Alabama; also in pheasants in the same areas. Human infection with western equine encephalomyelitis was not common in 1956.—*Pub. Health Serv., Jan. 4, 1957.*

Psittacosis was about 90 per cent more prevalent in man in 1956 than in 1955.—*Pub. Health Serv., Jan. 4, 1957.*

A woman, in Missouri, developed tularemia after being bitten on the finger by a sick cat which had frequently killed and eaten wild rabbits. She had handled no wild animals. A blood specimen from the woman was serologically positive for *Pasteurella tularensis* (1:640), and the cat was positive (1:40).—*Pub. Health Serv., Jan. 4, 1957.*

Anthrax was diagnosed in a stevedore after he had repacked a broken bale of wool (from Syria) unloaded from a steamship. His face was the only part of the body exposed and, although there were no skin abrasions, an itching developed below the right eye about an hour later and the next day there was a noticeable swelling. Smears and cultures contained *Bacillus anthracis*.—*Pub. Health Serv., Jan. 10, 1957.*

### Aftosa Diagnosed on Curacao

Foot-and-mouth disease (aftosa) recently appeared on Curacao, Netherlands, West Indies. On Jan. 24, 1957, an embargo was placed, by the U.S.D.A., on all susceptible livestock and their meat products from that island. This is the first appearance of aftosa in a previously noninfected Western Hemisphere area since 1954 when the disease was eradicated in Mexico.—*U.S.D.A., Washington, Jan. 30, 1957.*

## Surgical Management of Hypertrophic Pulmonary Osteoarthropathy in the Dog

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RECENTLY, interest has been focused on surgical therapy of dogs with hypertrophic pulmonary osteoarthropathy.<sup>1</sup> The surgical removal of a primary lung tumor failed to bring recovery and the animal was euthanized one month postoperatively because of disseminated visceral metastases.

At the University of Pennsylvania Veterinary Hospital, 9 dogs with hypertrophic osteoarthropathy, 8 of which had lung involvement, have been seen since July, 1950 (table 1). In the 2 which were treated surgically, the osteoarthropathy regressed for variable periods of time. However, both eventually died because of widespread metastases (case 1) or recurrence of the tumor (case 2).

### CASE REPORTS

**Case 1.**—On Nov. 14, 1952, a female Dalmatian, 8 years old, showed signs of listlessness, dyspnea, inappetence, and polydipsia of one week's duration and had a temperature of 105.4 F. She was alert and in good flesh. The lung sounds were dull in the ventral thoracic area with an increased vesicular murmur in the dorsal lung fields. The tentative diagnosis was bronchopneumonia and she was hospitalized.

After three days of treatment, with no change in condition, it was noticed that both carpal regions were diffusely thickened and the gait was stiff. Radiographs revealed periosteal changes typical of osteoarthropathy in the extremities (fig. 1). The proliferative lesions, which were prominent in the forelegs, presumably had only been present a short time. A huge mass with the density of bone (fig. 2) was located in the dorsocaudal region of the thorax, from the seventh to the eleventh intercostal space. On dorsoventral view, the mass was located in the region of the left diaphragmatic lobe. Several small masses of similar density were scattered throughout the lobes of the right lung. The clinical diagnosis was hypertrophic pulmonary osteoarthropathy associated with a large pri-

mary lung tumor containing bone or cartilage, which apparently had metastasized to other regions of the lung.

TABLE 1—Data on 9 Canine Cases of Hypertrophic Osteoarthropathy Seen at University of Pennsylvania, School of Veterinary Medicine, After July, 1950

Case No.	Breed	Age	Sex	Diagnosis*
1	Dalmatian	8 yr.	female	Chondrosarcoma of left diaphragmatic lobe with diffuse lung metastases.
2	Boston Terrier	14 yr.	female	Chondrosarcoma of left diaphragmatic lobe.
3	Boxer	8 yr.	male	Bronchogenic carcinoma of left apical lobe.
4	Boxer	9 yr.	male	Bronchogenic carcinoma of right apical lobe.
5	German Shepherd	7 yr.	male	Diffuse lung metastases from an epidermoid carcinoma of the left tonsil.
6	Mongrel	11 yr.	female	Diffuse lung metastases from a malignant "mixed" mammary tumor.
7	Mongrel	5 yr.	male	Pulmonary tuberculosis.†
8	German Shepherd	7 yr.	male	Vegetative valvular endocarditis (Streptococcus viridans).
9	Mongrel	4-5 yr.	female	Osteosarcoma of thoracic esophagus with diffuse lung metastases.

\*In all cases, necropsy with subsequent histological examination was carried out.

†Previously reported. Fielder, F. G., and Brodey, R. S.: What's Your Diagnosis? J.A.V.M.A., 127, (Sept. 15, 1955): 247-248.

On November 24, a thoracotomy was performed and the diaphragmatic lobe of the left lung, containing a large hard neoplastic mass, was removed. No other tumor nodules were found in the lung. The animal made a rapid recovery and was discharged six days after surgery.

The tumor, a round 7.5-cm. mass which surrounded the bronchus, contained much bonelike material. The histological features were identical to those of chondrosarcoma.<sup>2</sup> The center of the tumor was largely necrotic and contained calciferous deposits. Peripherally, well differentiated cartilaginous tissue was present together with cellular areas, some of which were extremely anaplastic.

Fifteen weeks after surgery, all signs of lameness had disappeared, the carpal swellings were no longer present, and the peri-

From the School of Veterinary Medicine, University of Pennsylvania, Philadelphia. Dr. Wind is now with the School of Veterinary Medicine, University of California, Davis.



Fig. 1—Preoperative lateral radiographs of the left radius and ulna of the Dalmatian (case 1) showing the soft tissue swelling and periosteal hyperplasia (arrows) characteristic of hypertrophic osteoarthropathy.

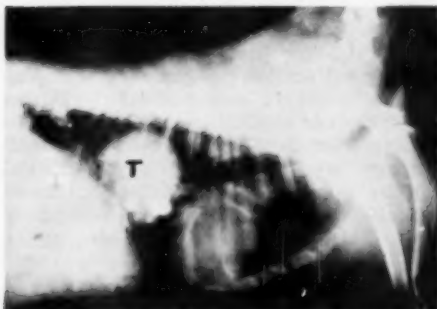


Fig. 2—Preoperative lateral radiograph of the thorax of the Dalmatian (case 1). Notice the large dense tumor (T) in the region of the diaphragmatic lobe.



Fig. 3—Radiographs of the left radius and ulna of the Dalmatian (case 1) taken three and a half months after surgery. Notice the absence of the soft tissue swelling and marked regression of the periosteal changes.

osteal changes had almost completely regressed (fig. 3). Coughing or dyspnea were not present, although radiographs showed that the metastatic lung tumors had increased both in size and number. However, three weeks later, she was re-examined because coughing, dyspnea, and lameness had recurred and the periosteal hyperplasia and soft tissue swelling of the limbs was more extensive than at the time of the initial diagnosis of osteoarthropathy. The proliferative changes in the hindlegs were much more prominent than those of the forelegs. Aside from the diffuse thickening, a localized painful bony mass had developed on the anterolateral aspect of the proximal portion of the left tibia. Radiographs also



Fig. 4—Lateral radiograph of the Dalmatian's left tibia (case 1), taken approximately six months after surgery, showing two chondrosarcomas (C) adjacent to the proximal tibia. Notice also the soft tissue swelling and diffuse periosteal proliferation (arrows) typical of hypertrophic osteoarthropathy.

revealed two circumscribed masses with the density of bone in the soft tissues adjacent to the tibia, thought to represent blood-borne metastases, and a large mass with a density similar to the lung tumors, which was palpable and movable, in the lower midabdominal region.

For the next two months, the bitch was examined clinically and radiographically every week. During this time, all symptoms increased in severity; the gait gradually became more stiff and painful, the legs thickened progressively and seemed to interfere with normal locomotion, the lesion on the left tibia rapidly became larger and more painful and the leg was held in con-

stant flexion. The abdominal mass enlarged (10 to 12 cm.) and caused a pronounced bulging of the abdominal wall. Due to the marked deterioration of her general condition, she was euthanized at the end of two months.

Radiographs taken just prior to euthanasia showed extensive periosteal proliferation and soft tissue swelling of the limbs and evidence of widespread erosion of the cortex of the bones. The tumors in the tibial region had tripled in size and had diffusely merged with the tibia. The metastatic lung tumors had become so large that they almost filled the pleural cavities.

**Necropsy Report.**—Neoplasms were present in the lungs, heart, liver, spleen, urinary bladder, kidneys, adrenals, and left tibia. The mammary glands were normal.

The bronchi and bronchioles were dilated and their walls were thickened. The lungs were studded with grayish white nodules, measuring 1.0 mm. to 4.5 cm. in diameter, all of which were hard and gritty when cut. The bronchial lymph nodes were slightly enlarged and contained several grayish, hard, gritty nodules.

The heart was pale and flabby with a hard gritty mass (2.5 by 2.0 cm.) at the junction of the left atrium and ventricle. Other nodules in the left ventricle were subendocardial (1 to 3 mm.), in the myocardium (1 mm.), and one epicardial (2 mm.). Several gritty nodules (0.5 to 1.0 mm.) were at the free margins of the mitral valve.

A mass in the middle third of the spleen measured 10 by 15 cm. and was so hard that it could not be cut. The right kidney contained numerous nodules (1.0 mm. to 4.5 cm.) and the left kidney, ten nodules (1.0 mm. to 1.5 cm.).

A 4.8- by 4.3-cm. mass in the wall of the urinary bladder projected into the lumen. Its mucosal surface was smooth, soft, and grayish pink. It consisted of a fibrous periphery and a gelatinous center.

Both adrenals were irregularly enlarged, difficult to cut, and had a well-circumscribed mass within the medulla. These lesions measured 1.0 by 0.6 cm. and 1.2 by 0.8 cm. with an outer hard, white, gritty layer and a soft, grayish red center.

The left tibia was tremendously enlarged with a grayish cartilaginous mass on its proximal portion, and the extremities were greatly thickened. All the long bones were covered by irregular deposits of periosteal



Fig. 5—Marked enlargement of the Boston Terrier's stifle and hock joints due to pulmonary osteoarthropathy (case 2). This picture was taken prior to surgery.

new bone. Histologically, the metastatic tumors varied considerably in their degree of cellular differentiation; however, all sections examined showed typical characteristics of a chondrosarcoma.

The clinical course and the size of the removed lung tumor suggests the lung as the primary site; however, this may be questioned because of the tibial involvement.

**Case 2.**—On Jan. 16, 1956, a Boston Terrier bitch, 14 years old, showed a bilateral swelling of the hock and stifle joints associated with marked stiffness and pain. She coughed occasionally and seemed to tire easily. All symptoms had been present at least two months.

She was active, although somewhat obese. There was marked bony thickening and soft tissue swelling of the hock and stifle joints and slight enlargement of the carpal joints. The stilted gait was apparent mostly in the pelvic legs. Auscultation of the thorax was negative. The third and fifth right mammary glands contained small neoplastic nodules.



Fig. 6—Preoperative radiographs (case 2) showing the sunburst appearance of the periosteal hyperplasia on the Terrier's hock areas (arrows). Periosteal proliferation is also present on the diaphyses of the tibiae and fibulae.

Radiographically, there were extensive proliferative periosteal lesions on the major long bones, which were most pronounced around the stifle and hock joints. In the latter, the "sunburst" appearance caused by the radiating bony spicules somewhat simulated osteosarcoma (fig. 6). Each stifle joint was greatly enlarged as a result of soft tissue swelling and periosteal lesions on the femoral condyles, tibial head and patella, and foci of calcification in the region of the quadriceps tendon just below the patella. Radiographic and fluoroscopic examination of the thorax revealed a large ovoid mass of uniform density, extending from the sixth to the ninth intercostal space in the region of the left diaphragmatic lobe. It was less radiopaque than the heart.

The hematological findings were normal. The clinical diagnosis was hypertrophic pulmonary osteoarthropathy associated with a primary lung tumor.

On thoracotomy, the following day, a large tumor covered by intact visceral pleura was found in the base of the diaphragmatic lobe and, since it was thought to be malignant, the entire left lung was resected.



Fig. 7—Radiographs taken approximately four months postoperatively (case 2) showing reduction of the periosteal hyperplasia and soft tissue swelling as compared with the preoperative films (fig. 6).

The tumor, a firm grayish slightly lobulated mass (5.0 by 4.4 cm.) which had eroded into the lumen of the bronchus, consisted of calciferous deposits mixed with areas of necrosis. The histological diagnosis was chondrosarcoma.

The bitch recovered rapidly and, in six

days, the soft tissue swellings around the joints reduced in size and the lameness was less severe. On the eighth postoperative day, when she was discharged, the gait was normal and the soft tissue swelling had almost disappeared, although the periosteal hyperplasia was unaltered radiographically. Two months after surgery she seemed in excellent health; the bony thickening of the hock and stifle joints was reduced.

Three months after the pneumonectomy, when the three caudal mammary glands which contained small tumor nodules were removed, radiographs revealed diffuse opacification of the left side of the thorax, making detail difficult to determine, and most of the periosteal changes were reduced about 50 to 60 per cent. The fuzzy borders seen on many of the active periosteal lesions in the early films were replaced by smooth regular borders, indicating these changes were undergoing regression. The periosteal lesions in the lateral hock areas (fig. 7), although reduced in size, were still quite prominent. The bitch made a quick recovery from the mastectomy and was discharged in six days. The mammary tumors were duct papillomas and fibroadenomas.

On May 20, she was re-examined because for a few days she had refused food, vomited occasionally, and was dyspneic. The oral mucosa was slightly cyanotic and there was a marked expiratory dyspnea. The

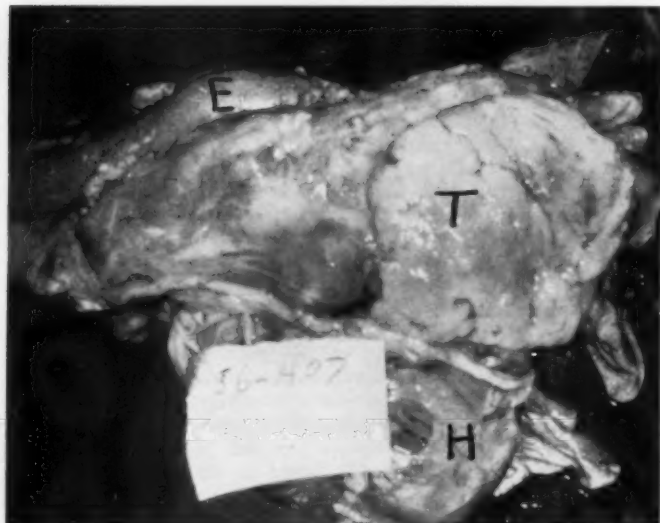
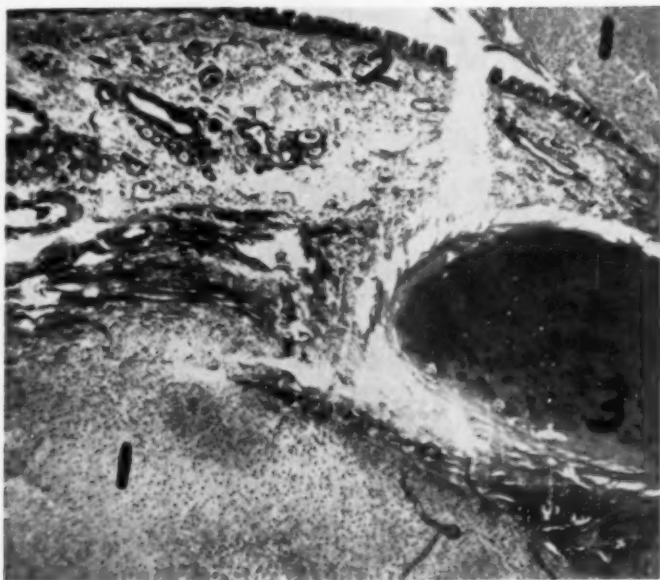


Fig. 8—Necropsy specimen of thoracic viscera (case 2) with recurrent chondrosarcoma (T) overlying the base of the heart (H) and showing the dorsal deviation of the esophagus (E).

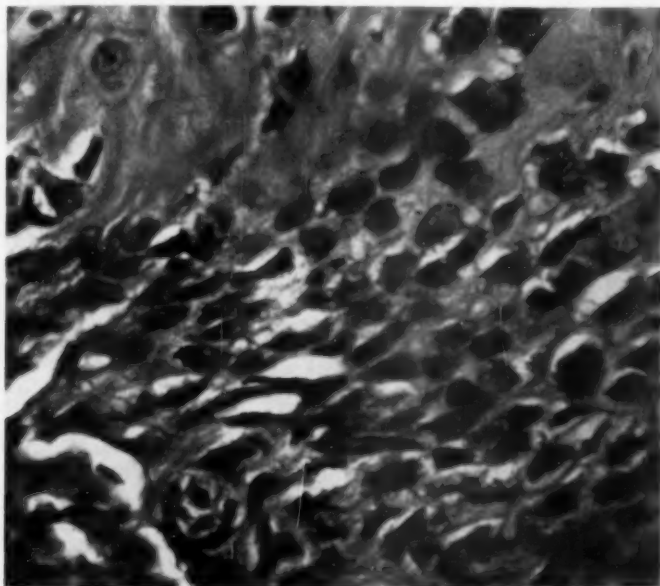
Fig. 9—Section showing relationship of the recurrent chondrosarcoma (1) to the bronchial mucosa (2) and the normal hyaline cartilage of the bronchus (3) (case 2).  
x 35.



lung sounds were slightly increased throughout the right thorax and there was moderate tachycardia. There was no soft tissue swelling of the extremities and, radiologically, the periosteal changes had undergone further regression. After aspirat-

ing 100 cc. of serous fluid from the left side, the thorax was examined radiographically. A large ill-defined mass in the left thorax pushed the trachea and esophagus dorsally and to the right and compressed the esophagus near the base of the

Fig. 10—Section showing tumor cells of anaplastic area within the chondrosarcoma. (case 2). x 475.



heart. Distal to this compression, the esophagus and the stomach were distended with gas.

Although the dyspnea was temporarily relieved by the thoracentesis, it increased and death occurred on May 26.

**Necropsy Report.**—In the thorax, a large, firm, grayish white, oval tumor (10.5 by 7.0 by 4.5 cm.), on the left side, extended over the base of the heart and displaced the trachea and esophagus to the right (fig. 8). The stump of the left bronchus could be traced for a distance of 1.5 cm. before its lumen became obliterated by the neoplasm. Cartilaginous tissue was mixed with areas of softer tissues. Histologically, the recurrent chondrosarcoma appeared to originate from the bronchial wall at the site of resection (fig. 9).

Small, grayish white, gritty nodules were observed on the mediastinum, the thoracic surface of the left diaphragm, and the left parietal pleura; the largest (2.0 by 2.5 cm.) was on the pleural surface of the eighth rib near its costochondral junction. There was marked congestion and edema of the right lung and severe adrenal cortical hyperplasia.

The long bones were macerated and studied grossly and radiographically. Some periosteal thickening was apparent on the proximal and distal portions of the tibiae and fibulae and, to a lesser extent, on the distal radii and femurs.

#### DISCUSSION

In each case, clinical and radiographic signs of osteoarthropathy decreased following surgical removal of the primary lung tumor. Lameness and soft tissue swelling disappeared within the first eight postoperative days. The periosteal changes regressed at a slower rate, although they were greatly reduced after three and a half months (case 1) and four months (case 2).

However, the metastatic chondrosarcomas in the lung were enlarging while the osteoarthropathy was regressing. Apparently, it was necessary for the lung tumors to reach a certain point of development before influencing the mechanism leading to osteoarthropathy. The effect of their presence on the rate of periosteal regression remains speculative.

In case 2, the lack of exacerbation of the osteoarthropathy probably was related to the absence of neoplastic involvement of the remaining lung.

In many animals, the signs of osteoarthropathy appear long before respiratory symptoms are apparent. In case 6 (table 1), a malignant mixed tumor of the fourth and fifth mammary glands was removed and, for two years, the bitch appeared normal. However, in the third year, lameness appeared associated with progressive enlargement of the carpal and tarsal joints. She was treated for what was thought to be polyarthritis. Although respiratory symptoms were absent until shortly before death, at necropsy, diffuse pulmonary metastases were found.

In man also, osteoarthropathy is often misdiagnosed as multiple arthritis or occasionally as acromegaly.<sup>3</sup> The presence of multiple articular and long bone thickening should immediately alert the clinician to the probability of osteoarthropathy associated with an intrathoracic lesion. The reversibility of pulmonary osteoarthropathy should encourage the veterinarian to make an accurate diagnosis so that resection of the causative lesion can be attempted in selected cases.

#### ADDENDUM

On Nov. 10, 1956, a male Boxer, 8 years old, was presented to the clinic and a diagnosis of hypertrophic osteoarthropathy was made. Radiological examination of the chest revealed a large mass in the region of the right cardiac lobe.

A lobectomy for the removal of a large lung tumor located in the right cardiac lobe was performed on the day of admission. An enlarged tumorous bronchial lymph node was also resected and small metastatic foci (1 to 3 mm.) were noticed in the remaining lobes of the lung. The histological diagnosis was primary pulmonary adenocarcinoma. The dog made a rapid recovery and, six days postoperatively, the lameness and soft tissue swelling of the limbs had completely disappeared. This case will be closely followed and reported on more fully at a later date.

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# The Toxicity of Organic Phosphorus Insecticides to Livestock

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A NUMBER of organic phosphorus compounds with excellent insecticidal properties have been developed. The earlier ones were highly toxic for mammals so their use was limited to a few plant crops. Many of the later compounds are much less toxic for mammals. Organic phosphorus insecticides are widely used to control plant pests, flies, and parasites of small animals; eventually, some will be used to control parasites of livestock. Interest in these compounds has increased due to the development of resistance in parasitic arthropods to the chlorinated hydrocarbon insecticides. In Africa, South America, and Australia, the resistance of ticks to chlorinated hydrocarbons is high and extensive efforts are being made to use the phosphorus compounds.

In the United States, the principal exposures of livestock to organic phosphorus insecticides are through consumption of deliberately or accidentally treated forage, spillage, carelessly disposed containers, or the consumption of baits. Our studies have included spraying, dipping, and oral administration.

## EXPERIMENTAL DATA

The animals used in these tests were in good health and in good range condition. They consisted of locally purchased grade cattle, 1 year old or over, grade dairy calves, and grade Rambouillet and Delaine sheep.

Sprays and dips were prepared from water-wettable powders or emulsifiable concentrates diluted with water on an equal weight basis and were used to completely wet the animals to the skin.

Oral administration was with a dose syringe, using emulsions or suspensions prepared from the concentrates used for spraying.

A compound is classified as toxic when it causes any observable deviation of the animal's behavior. The  $LD_{50}$  for these com-

pounds would be much higher, perhaps even higher than some of the doses classified as lethal. However, this classification is used to avoid any intoxication from normal usage. (A comparison of the toxicities of these compounds with the better known chlorinated hydrocarbon insecticides can be made.<sup>2</sup>)

The data presented here (tables 1, 2) represent an incomplete picture but serve to indicate the relative toxicities of the compounds.

## SIGNS OF TOXICITY

Usually the signs of toxicity are associated with impairment of the enzymatic destruction of esters of choline. An exception is O, O-dimethyl-O-2, 4, 5-trichlorophenyl phosphorothioate (Dow ET-57) which has this action but has a predomi-

TABLE 1—Summary of the Results of Oral Treatment of Cattle and Sheep with Various Organic Phosphorus Insecticides

Chemical	Animal	Dose (mg./kg.)		
		Lethal	Toxic	Nontoxic
Guthion (Bayer 17147)	Calves	25.0	0.5	0.1
	Sheep	—	25.0	12.5
	Calves	1.5	0.5	0.25
	Sheep	75.0	50.0	25.0
Parathion	Sheep	20.0	—	10.0
	Calves	—	1.0	0.5
	Sheep	—	—	10.0
	Sheep	20.0	—	10.0
Diazinon	Calves	10.0	1.0	0.5
	Sheep	—	25.0	10.0
	Sheep	—	30.0	20.0
	Sheep	—	—	—
American Cyanamid 12009	Sheep	10.0	5.0	—
	Calves	10.0	5.0	—
	Sheep	—	10.0	—
	Calves	—	10.0	—
Hercules 528	Sheep	—	25.0	10.0
	Calves	—	10.0	5.0
Pirazinon	Sheep	—	100.0	50.0
	Calves	—	200.0	100.0
DDVP	Sheep	—	20.0	10.0
	Calves	—	100.0	50.0
Dipterex (Bayer L 13/59)	Sheep	—	20.0	10.0
	Calves	—	100.0	50.0
Malathion	Sheep	—	25.0	—
	Calves	—	100.0	50.0
	Sheep	150.0	100.0	50.0
	Calves	—	—	—
Dow ET-15	Sheep	—	100.0	—
	Calves	—	—	100.0
American Cyanamid 4124	Calves	—	25.0	5.0
	Calves	50.0	—	25.0
Chlorthion	Calves	—	—	50.0
	Calves	—	—	25.0
Geigy 25039	Sheep	—	—	25.0
	Calves	—	—	25.0
Dow ET-14	Calves	—	100.0	50.0
	Sheep	—	—	100.0
Dow ET-57	Calves	—	125.0	100.0
	Sheep	—	400.0	100.0

From the Animal Disease and Parasite Research Branch, ARS, U. S. Department of Agriculture, Kerrville, Texas. Presented before the Section on Research, Ninety-Third Annual Meeting, American Veterinary Medical Association, San Antonio, Texas, Oct. 15-18, 1956.

TABLE 2—Summary of Results of Spray and Dip Treatments of Cattle, Sheep, and Goats with Various Organic Phosphorus Insecticides

Chemical	Animal	Percentage of insecticide in spray or dip		
		Lethal	Toxic	Nontoxic
Parathion	Calves	0.02	0.01	—
	Sheep	1.0	—	—
	Goats	1.0	—	0.1
Pirazinon	Calves	—	0.05	—
EPN	Calves	0.25	0.05	0.025
Diazinon	Calves	0.25	0.1	0.05
Bayer 18510	Calves	—	0.1	—
Bayer 16/599	Calves	—	—	0.1
Guthion	—	—	—	—
(Bayer 17147)	Calves	0.5	0.25	0.1
Bayer R 1656	Calves	0.5	0.25	0.1
Hercules 528	Calves	0.5	0.25	0.1
Bayer 21/199	Yearlings	—	—	0.5
	Calves	0.75	0.5	0.25
Bayer 19641	Cattle	—	—	2.0
	Calves	1.0	—	0.25
Malathion	Calves	1.0	—	0.5
	Turkeys	—	—	2.0
NPD (DuPont)	Calves	—	—	5.0
DDVP	Cattle	—	—	0.5
Dipterex	—	—	—	1.0
(Bayer L 13/59)	Calves	—	—	1.0
Geigy 25039	Calves	—	—	1.0
Bayer 19596	Calves	—	—	2.0

nating syndrome similar to chlorinated phenols.

Poisoned animals generally first show excessive salivation. The saliva flows abundantly and approaches the consistency of water. The animal then breathes with the mouth open and with greatly exaggerated respiratory movements. As the respiratory effort increases, the animal walks stiff-legged and wanders about restlessly. Fasciculations of all skeletal muscles are present. Eventually, exhaustion forces the animal to lie down. As death approaches, there are pulmonary râles and the animal grunts softly. Death appears to occur by suffocation. Only with the highest doses have convulsions been seen.

Dow ET-57 apparently has two modes of action. The first signs include depression, pronounced muscular weakness, incoordination, and prostration, usually accompanied by diarrhea. These appear within 24 hours of exposure and may continue for several weeks. Moderate poisoning lasts one to two weeks. At high dosages, some salivation and dyspnea may be seen five to eight days after exposure.

#### CHOLINESTERASE

All of the compounds discussed in this paper inhibit cholinesterase. Their effects upon the erythrocyte cholinesterase (there is virtually none in the plasma) of sheep

and cattle has been followed with the electrometric method<sup>1</sup> primarily to establish a guide for diagnosis. Unfortunately, diagnosis by this means is not easy since there is wide variation between individuals. When the control value is not known, the finding in a single case lacks significance unless extremely low or well within the normal range; therefore, at most, a cholinesterase determination in a single case may be confirmative only if extremely low.

While the studies are not complete, a number of compounds given daily in small doses will progressively depress cholinesterase to essentially zero; yet, no objective symptoms will be displayed. In acute cases, the same value would be accompanied by severe signs of poisoning.

Blood cholinesterase determinations are used as a guide. It is possible for animals to be demonstrating severe symptoms while the blood cholinesterase is relatively high. It is also possible to have an essentially zero reading without apparent signs of poisoning.

#### LESIONS

In acute poisoning, the lesions found at necropsy are never outstanding and never pathognomonic; in many cases, the findings are entirely negative.

The lesions when they appear, may be hemorrhages of varying sizes on the heart, lungs, or gastrointestinal tract which are not consistent in their location. The lungs may be congested and are often edematous and heavy. Frothy exudate may be present in the bronchi and trachea. If the animal has been affected over a prolonged period, pneumonia may be observed. The lungs of several animals, which breathed as if they had pneumonia, were free of lesions at necropsy.

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Although about half of the lymphoid tissue in a rabbit's body is located in the appendix, several dozen rabbits were apparently unaffected by appendectomies.—*Nature*, Oct. 6, 1956.

## Fescue Foot Lameness in Cattle—Some Observations on the Disease in Virginia

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FESCUE LAMENESS of cattle is a noninfectious disease characterized by a dry gangrene of the feet and tail. It is believed to be due to the consumption of tall fescue grass (*Festuca arundinacea*) or some of its varieties.

The condition was first described, in 1949, in New Zealand.<sup>1</sup> It was reported in western Colorado, in 1952,<sup>2</sup> and in Kentucky, in 1953,<sup>3</sup> when cattle were grazing on cultivated Kentucky fescue 31. In 1956,<sup>4</sup> it was experimentally produced, in Colorado, while cattle were grazing on fescue or while eating fescue hay. No selenium was present but an alkaloid similar to ergot was found in the tall fescue grass.<sup>5</sup>

During the past year, an opportunity occurred to examine more closely some cases of lameness in cattle which were suggestive of fescue lameness.

An equal number of Shorthorn, Aberdeen Angus, and Hereford heifers, all coming 2-year-olds, were purchased by the experiment station to be wintered and prepared for a cross-breeding experiment. These 119 animals had been selected from the best sources in the state, 4 or 5 from each source, to get as much uniformity as possible when the crosses were made. They were pastured at a substation prior to arrival at the station.

Three pastures had been used at the substation: (1) A 40-acre tract had been seeded to Kentucky fescue 31 and lespedeza in March, 1953; (2) an 18-acre tract was seeded with spring oats and fescue; and (3) a small 3-acre lot contained a mixed pasture growth but no fescue. The rate of seeding for fescue in all tracts was 10 lb. per acre. The seed was of a strain produced locally for nine years. Hay had been harvested from tract 1 in the fall of 1954; hay was harvested from both tracts in June, 1955. Sixty heifers were turned into pastures 1 and 2 on Nov. 8, 1955. The first sign of lameness, observed in the first week of December, was in a white heifer. By

December 13, she had become so lame it was necessary to remove her to the barn. All were removed from the pastures on December 19 and prepared for shipment to this station. Many in both lots had suffered from so-called "foot rot"; the white heifer had been affected in June, 1955.

The weather had been cold during all of the period the animals were grazing on fescue, and the fescue was thoroughly frosted. Lameness was first seen after a week of cold, rough weather when the ground was hard and uneven. After one snowfall, a small amount of rough clover hay had been fed, the only feed given other than that provided by the pastures.

The animals were shipped some 90 miles by truck during the last week in December, when it was bitterly cold. On arrival, 2 were acutely ill and were treated for pneumonia, the most obvious clinical condition, although both showed lameness and stiffness as well as excess salivation. Due to its common occurrence, during this period, they were carefully checked for mucosal disease but showed no evidence of it. Since it was possible that they had so-called "shipping fever," the balance of the herd was examined. The heifers were allotted to two pastures. They ate poorly and refused grass silage. In one lot of 75, 9 were lame in the hindfeet and 1 Aberdeen Angus was lame in all feet. In the lot of 42, 1 Aberdeen Angus was lame in all feet and 8 others, Herefords or Shorthorns, were lame in the hindfeet—about 60 per cent in the left hindfoot. There did not appear to be any predominance of lameness in the white feet.

The cattle had been without water for some time before shipping and, on arrival, had consumed large quantities of water from a tank in which the ice had to be broken.

The white heifer was observed to have a strip of dry skin about 2 inches wide encircling the coronary area of the left hindfoot.

When examined again, three days later, 6 were lame in the lot of 75 and 10 in the

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lot of 42. Lameness was again apparent in all feet of several animals, including the Aberdeen Angus mentioned above, but most of the lameness was in the hindfeet, especially in the left hindfoot. Those lame in all feet appeared to have laminitis, believed to be caused by excessive consumption of cold water. The sensitiveness seemed to be in their feet and the hoofs felt hot.

Four days later, 15 head, taken from both lots, were removed to a barn where they could be examined thoroughly. All were lame to a varying degree. The white heifer was also hospitalized for closer observation. During this procedure, she lost the strip of dry, gangrenous skin at the coronary band, leaving a circular, raw, bleeding area. She was very lame. On the following day, Jan. 9, 1956, 12 of the 15 animals (3 Aberdeen Angus had recovered) were placed in a chute and carefully examined. The results are shown (table 1).

The mouth lesions were those which would be considered typical of fluorine poisoning as far as the gums were concerned. Animals with open, infected fissures or sores on the feet were given local treatment and antibiotics parenterally. The 3 Aberdeen Angus heifers that had been tender in all feet were now sound. By the first week in February, two months after she was first noticed, only the white Short-

horn and 2 Herefords remained on the sick list. Both Herefords were lame in the hindlegs, with foul-smelling pus being discharged around the skin-hoof junction. One of these (No. 45) had previously been listed as lame but with no visible lesions. In these animals, the hoof proper appeared to be separating from the corium. They recovered with no permanent disability when given local treatment and massive doses of antibiotics.

Many of the Herefords also appeared to have a dermatitis in the white area of the back, with scaling and peeling of the superficial layers of the skin.

By early March, the white heifer had lost both digits of the left hindfoot. She was walking on the exposed distal portion of the second phalanges and had lost weight. She was killed for necropsy. No other important lesions were observed. The hindlegs were preserved in formalin and were then carefully dissected. No evidence of thrombosis was seen; however, thrombosed areas may have been destroyed when the digits were sloughed or when she walked on the stumps.

#### DISCUSSION

It may be surmised that the animals which showed tenderness in all feet were suffering from laminitis but it seems reasonably certain that the white heifer was suffering from fescue lameness. The others, however, may have had mild attacks of fescue lameness, sufficient to cause some separation of the hoof from the corium, allowing secondary infection to enter. This may have been aggravated by recrudescence of foot rot. The symptoms and history would cover any, or all, of these conditions in the different animals.

The possibility of photosensitization can not be eliminated. That color could be a factor is suggested by the rarity and type of lameness in the Aberdeen Angus heifers. The condition in cattle in South Africa is called "congenital porphyria" or "pink-tooth." However, this would not account for the mouth lesions.

Fescue lameness, photosensitization, and the weather may all have been factors. At the time the animals became ill, the weather was rough, the ground was frozen, and the fescue had been thoroughly frosted. The pasture was not all fescue but the fescue had resisted the dry, cold weather

TABLE 1—Results of Examination of Cattle, Jan. 9, 1956\*

Animal (No.)	Breed	Findings
86	Red Shorthorn	Interdigital fissure, bad odor from the right hindfoot and vesicles on the lips.
48	Roan Shorthorn	No visible foot lesions but necrotic areas near base of the teeth.
102	White Shorthorn	Fluorosis-like lesion in the mouth but no visible foot lesions.
63	Hereford	Lame but no lesions.
50	Hereford	Heat in all hoofs and scaly lesions in both hindfeet with spots on the coronary area of both hindfeet and granulation taking place under the scales.
91	Roan Shorthorn	Heat in one foot and sores on the coronary area.
64	Hereford	Lame but no visible lesions.
110	Roan Shorthorn	Fluorosis-like lesions in the mouth.
82	Roan Shorthorn	Dry, scaly lesions on one foot and fluorosis-like lesions in the mouth.
108	Shorthorn	A fissure on the coronary area of the right hindfoot and scars in the mouth.
109	Shorthorn	Lame but no visible lesions.
45	Hereford	Lame but no visible lesions.

\*Three of the 15, all Aberdeen Angus, had recovered.

better than the other grasses had. Most cases of photosensitization have been reported to show bilirubinemia,<sup>6</sup> which is considered to be evidence of liver damage, and most photosensitization is considered to be hepatogenic in origin. However, the white heifer and one other animal were checked for bilirubin and both gave a negative reaction.

#### SUMMARY

What is considered to have been fescue lameness in cattle occurred in a group of 119 heifers, about an equal number of Shorthorns, Herefords, and Aberdeen Angus. Of 16 affected, only 3 were Aberdeen Angus and all recovered quickly. All of the others, except 1 of 2 white Shorthorns, also recovered. About two months after she became ill, the digital extremities of a hindfoot of this heifer were sloughed. She was killed for necropsy but no other lesions were found. The exact cause was not established.

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*Can Tubercle Bacilli Form Spores?*—A study of the internal anatomy of avian tubercle bacilli, by means of an electron microscope, suggests that under certain conditions the avian tubercle bacillus produces spores.—*Nature*, Sept. 8, 1956.

*Mucosal Disease in a Deer.*—A syndrome was found in white-tailed and mule deer, in North Dakota, which in many respects resembled mucosal disease of cattle. The disease was experimentally transmitted to other deer and to antelope. Also, mucosal disease of cattle produced typical signs and lesions when transmitted to deer.—*Vet. Med., Aug.*, 1956.

#### Notes on Control of Hog Cholera

Three methods of immunization against swine fever have been used successfully in Switzerland but each had its disadvantages. The virulent virus, used with serum, was excreted for at least four weeks after vaccination. The crystal violet vaccine took two to three weeks to develop immunity. The lapinized vaccine conferred a solid immunity after seven days but produced leukopenia which rendered animals susceptible to bacterial diseases. The crystal violet vaccine was considered the method of choice.

• • •

Lapinized vaccine produced a quick and lasting immunity to swine fever, in France, but the virus was highly pathogenic for young pigs, especially the unborn. It is recommended that this vaccine be limited to pigs on recently infected farms. It should not be used on pregnant sows.

• • •

Lapinized vaccine is preferred, in China, to crystal violet vaccine because it is cheaper and confers a quicker and more solid immunity. When the virulence of the lapinized virus was tested by passage in pigs, there was no mortality at the fifth passage, a few deaths between the sixth and ninth passages, and all pigs died at the tenth passage.

• • •

An epizootic of swine fever (hog cholera) was controlled, in Central Germany, by mass vaccination with crystal violet vaccine. A fifth of the 25,000 swine in the area had died or been destroyed because of the disease. A few weeks after vaccination, the region was free of cholera and has remained so. This vaccine, with veterinary police measures, is considered an effective method of control.—*Vet. Bull., Dec.*, 1956.

*Trembling in Newborn Pigs.*—The cause of trembling in newborn pigs is not known. The results of selective matings indicate that it is not hereditary. The tremors are present at birth and seem to affect all of the muscles except those innervated by the cranial nerves. If the pig survives, recovery is usually complete in four to eight weeks. While histological studies are incomplete, the nerve cells appear to be normal.—*Proc. Roy. Soc. Med., Dec.*, 1956.

## Filarial Dermatitis of Sheep

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FILARIAL dermatosis of sheep is a skin malady resulting from the presence of the adult bloodworm, *Elaeophora schneideri* (Wehr and Dikmans, 1935), in arterial blood vessels and of its microfilarial or immature form in the skin of the same sheep host.<sup>1</sup> The presence of this microscopic larva (fig. 1) in the skin causes lesions on



Fig. 1.—Microfilaria of *Elaeophora schneideri* recovered by the maceration technique. x 500.

the head (fig. 2, 3), feet (fig. 4), and abdomen. This infection is apparently contracted while the sheep are grazing on summer ranges at elevations above 6,000 ft. in some mountain localities of New Mexico, Colorado, and Arizona. Presumably, the lesions do not become evident until the year after the sheep have contracted the infection. The youngest sheep observed with a head lesion was 21 months old, but most are more than 2 years old.

This disease was first reported by the author<sup>1</sup> in 1938 but had been observed by flock owners in New Mexico since 1932. They referred to the affected animals as "sore-headed sheep." They segregated the affected animals the first year and treated them repeatedly with local applications of germicides without beneficial results. A significant increase in the number of sheep showing skin lesions the following year prompted the owners to enlist the aid of

veterinarians in the U.S. Department of Agriculture.

The areas in New Mexico where sheep showing skin lesions apparently originate are confined to high grazing areas in the Mogollon Mountains in the southwestern part of the state. The common range practice is to trail sheep flocks to summer mountain ranges, at elevations of 7,000 to 10,000 ft., about April 1 of each year. About October 15, they are trailed back to their winter ranges in the foothills, at elevations of less than 6,000 ft. in Catron and Socorro counties.

After 1933, the bloodworms responsible for the skin lesions were found on necropsy of sheep grazing at the higher elevations of Apache, Navajo, Coconino, and Yavapai counties in Arizona, and in Montezuma, La Plata, and Archuleta counties in Colorado where the grazing practices are similar to those in New Mexico. Filarial dermatosis was also reported<sup>2</sup> in a flock of sheep that had spent the summer on a range in Plumas County, California, near the summit of the Sierra Nevada Mountains, and the causative threadlike worms were found in 3 ewes reported to have been shipped from Lake County, Oregon, into California for slaughter.

The disease was found for the first time, in 1951, in 3 sheep of a flock near Kalispell, Mont.<sup>3</sup> It is uncertain whether they had been shipped in or were native Montana sheep.

Several cases of this disease in sheep were observed near Pender, Neb., but the animals involved apparently had been shipped into the state.

A condition resembling filarial dermatosis of sheep was observed in North Dakota in 1955.<sup>4</sup> The infected sheep had recently been shipped into the state and were causing authorities much concern.

The first specimens of the bloodworm, *E. schneideri*, collected from deer was in 1934 in Beaver County, Utah.<sup>5</sup> The threadlike bloodworms were obtained from the cut surface of the head of a mule deer killed within the preceding 24 hours.

Several specimens of *E. schneideri* were recovered from the arteries of a buck deer

Dr. Kemper is chief veterinarian, Sheep Sanitary Board of New Mexico, Albuquerque.



Fig. 2—Extensive skin lesion of filarial dermatosis of sheep involving poll, face, nostrils, and lips.



Fig. 3—Circumscribed, encrusted, quiescent head lesion of filarial dermatosis of sheep.

on Vancouver Island, B. C., in March, 1940.<sup>7</sup> Subsequently, 1 immature female of this worm was taken from a coast deer on the same island. No visible symptoms arising from the presence of this bloodworm were observed.

In 1952, a high incidence of this bloodworm was found in deer in two areas in Lake and Tehama counties, California.<sup>8</sup> Collection records of the laboratory of the California Division of Fish and Game show that this same worm has also been found in deer in Plumas, Sierra, Tuolumne, and Inyo counties, the incidence ranging from 4 per cent in Tehama County to 44 per cent in Inyo County. These records, however, may not give an accurate picture because sampling has not been general throughout the state.

#### ADULT BLOODWORM

Several specimens of *E. schneideri* were received from Arizona\* in May, 1953. They were from 3 of 5 mule deer taken in Yavapai County at an elevation of 4,000 ft. The deer had no noticeable lesions.

The adult bloodworm, *E. schneideri*, was first found in sheep by the author in New Mexico in March, 1934.<sup>9</sup> These threadlike

worms were consistently present in the larger arteries of sheep manifesting head, foot, or abdominal skin lesions, which suggested that they were in some manner associated with the lesions. The worms were found unattached in the larger arteries and in the left ventricle of the heart. When exposed to the air, they immediately assumed the shape of a coiled watch spring and recoiled after being forcibly straightened out to be measured.

To recover the worms, it was necessary to cut the sheep's throat, as for slaughter, and let all of the blood pass through a piece of ordinary window screen; they could be



Fig. 4—Foot lesion of filarial dermatosis of sheep, resembling a clubfoot.

\*The specimens were contributed by A. W. Yoder and W. G. Swank, Wildlife Restoration Division, Arizona Game and Fish Commission.

recovered from other arteries only by opening the vessels. The parasites were then observed in a straightened position.

The worms are glassy white in color, and are tapered to a point at both ends. They measure from 6 to 12 cm. (2 2/5 to 4 4/5 in.) long, the males being about one half as long as the females. The diameter varies from 0.74 to 0.83 mm. (1/34 to 1/30 in.) at the center of the body. The male is spirally and tightly coiled at the posterior extremity and gradually tapered to a point at the anterior end, while the female is gradually tapered to a point at both extremities. Males and females are often found lying in pairs in the arteries. No injurious effects have been observed from the presence of the adult bloodworms in the arteries.

When head lesions were present, the adult worms were usually found in the larger arteries supplying the blood to the head. When foot lesions were present, the worms were commonly found in the large arteries of the affected leg. The worms were not found in the arteries supplying blood to the area where the abdominal or chest lesions occurred. However, this can not be considered as conclusive of their absence because the parasite might easily have been overlooked during routine examination.

#### BLOODWORM MICROFILARIAE

Microfilariae (immature forms) were first found by the author in head lesion tissues in September, 1937. They were presumed to be the progeny of the adult bloodworm, *E. schneideri*, since no other threadlike worms are known to occur in the arteries of sheep in this country.

The microfilariae measured approximately 0.017 by 0.27 mm. and were virtually the same size and shape as those reported<sup>8</sup> in the egg membrane expressed from the uterus of the adult female bloodworm. The microfilariae, when stained by the Fulleborn method,<sup>9</sup> were bluntly rounded at the head end and were tapered to a point at the posterior end. The anterior end was devoid of granular staining material for a short distance, but the remainder of the body structure appeared granular, with the exception of a faintly brownish, uniform-appearing mass located in the middle third of the body and resembling, in outline, three flattened united circles.

The presence of these microfilariae in all skin lesion tissues, and their absence from nearby unaffected skin, established them as the cause of filarial dermatosis of sheep.

The eggs removed from adult females were devoid of a shell but were enveloped in a thin membrane. The eggs are presumably carried in the arteries to the capillaries, where they are trapped

and where the released microfilariae become lodged.

The only other known species of this genus is the *Elaeophora poeli* (Vryburg, 1897), which is found with its head end usually embedded in nodules inside the aorta of water buffalo and zebu cattle in Asia. It is more than twice the length of the American bloodworm, *E. schneideri*.

#### COURSE OF THE INFECTION

The presence of the microfilariae of *E. schneideri* in the skin serves as an irritant that creates an intense itching. The sheep rub and scratch the involved areas, causing mechanical destruction of tissues. The laceration of the head lesions with the hindfoot, especially when a lesion becomes active, produces a raw, bleeding, inflammatory area of a granular nature, infiltrated with leukocytes and containing numerous small pus abscesses within the skin. The head lesions are most often observed, in the region of the poll, as small circumscribed areas varying from 5 to 10 cm. in diameter. However, the lesions may extend over the face and involve the nostrils, lips, and mouth. Lesions were observed inside the pharynx, nostrils, and mouth, in addition to a head lesion, in 1 sheep in Colorado.

Where a hindfoot is similarly involved, it is the one commonly used in scratching the head lesion. Foot lesions usually extend from the coronary band to above the pastern joint and resemble a clubfoot.

Lesions on the abdomen may or may not be in the general location where an infected foot could come in contact with the body when the sheep was lying down. However, the mere contact of a foot with a head lesion, or an infected foot in contact with the abdomen, is not believed to be the method of transfer of microfilariae from affected to healthy skin on the sheep because all other threadlike worms are known to require an intermediate host in their life cycle.

There are recurrent quiescent periods in which the irritation temporarily subsides. They may last from one to several days and permit the formation of raised encrustations over the affected area. These crusts are later scratched off, exposing a raw bleeding surface. The periodic cycle occurs over a period of seven months or longer and, with each succeeding cycle, the area of the lesion increases slightly. The amount of irritation in a lesion is commensurate with the number of adult worms in the corresponding arteries. The recurrent periods

of activity in the lesion are believed to be produced by additional new generations of microfilariae.

This recurrent irritation frequently stimulates a rudimentary horn growth on otherwise hornless Rambouillet ewes.

When all activity in a lesion permanently subsides, which may eventually occur, the lesion develops dry, grayish, flaky skin scales which sometimes resemble fungal (ringworm) infections. When a lesion has completely healed and the skin again assumes a normal appearance, new wool grows in the area.

No visible symptoms have been associated with the presence of the adult worms in the arteries of either sheep or deer. Furthermore, no skin lesions have been reported in deer in which the adult worms were found. This would lead to the speculation that deer may possibly be the primary host of this parasite, and that they may serve as the reservoir of infection for sheep. If so, sheep may be considered as an accidental host.

Since Filariidae require an intermediate host in their life cycle, it must be assumed that transmission of the disease occurs through the agency of blood-sucking insects, possibly from deer to the sheep, or from sheep to sheep.

Usually, not more than about 1 per cent of the sheep in infected flocks show lesions. However, greatly increased numbers might appear under favorable conditions.

#### DIAGNOSTIC METHODS

Filarial dermatosis of sheep presents a problem of diagnosis. Finding the adult worm in the arteries at the time of slaughter is the diagnostic method of choice. However, if they are not found, a search should be made for the immature forms in skin lesions by the maceration technique. This method, which has proved practical, consists of finely shredding the skin lesion tissues, placing them in warm physiological salt solution for two hours, and centrifuging to congregate the microfilaria at the bottom of the tube. The residue should reveal, under a compound microscope, several active, motile larvae.

This must be done on the day of necropsy. If not, the skin lesions may be fixed in 10 per cent formalin solution and later submitted to a pathologist for examination.<sup>11</sup> Making deep skin scrapings from the lesion

has not always been satisfactory but may be attempted if circumstances demand it.

#### TREATMENT

The following antimony compounds<sup>12</sup> have proved effective in treating this condition: trichicide, 1 oz. intravenously at weekly intervals for six consecutive weeks, destroyed the adult worms in the arterial blood stream, as well as the immature forms in the skin lesions; fudadin in single doses of 1 1/3 oz. intramuscularly resulted in the healing of skin lesions, but 2 doses at an interval of ten days are recommended for consistent results (both the adult worms and immature forms were destroyed by this treatment); anthiomaline (a French product, each oz. containing 4 1/2 gr. of antimony) in a single dose of 1 1/6 oz., either intramuscularly or intravenously, usually resulted in healing of the skin lesions and destruction of adult worms in the arteries—two such single doses at an interval of ten days is recommended for consistent results; tartar emetic, eight 1-oz. injections (1/2 oz. in each hindleg) proved effective and economical; daily doses of arsenamide (1/2 oz. of 1% solution to each 100 lb. of weight) intramuscularly for 15 consecutive days effected the destruction of immature bloodworms in the skin and the adult threadworms in arterial blood, resulting in the healing of the skin lesions within two weeks after completion of treatment; caracide, 4 1/2 gr. to each 10 lb. of body weight, incorporated in the feed for 15 consecutive days, resulted in healed skin lesions three weeks following treatment—larger doses were not well tolerated.

The too rapid destruction of the adult bloodworms may account, in part, for the harsh effects of some of the above mentioned chemicals.

With the use of these chemicals, an occasional loss of an animal may be expected. All affected sheep should be fed a high protein diet of grain and alfalfa hay a few days before and during the entire time of the treatment. This will materially reduce the losses incident to the treatments and will also maintain the affected sheep in better physical condition while they are undergoing treatment.

Some ranchers killed all visibly affected sheep for several successive seasons, but new cases were observed each season when the sheep returned to their winter range.

Since the life history of this bloodworm is still unknown, medicinal treatment appears to be the most effective means of control. Treatments are, however, only palliative measures.

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### Uremia Associated with Renal Calculi and Nephrosis in a Dog

A female Boxer, 10 years old, had been in declining health for three years. The predominant signs had been a gradual

A lateral radiograph of the abdomen (fig. 1) showed an opaque shadow in the area of the kidney.

Because of the poor prognosis, eutha-

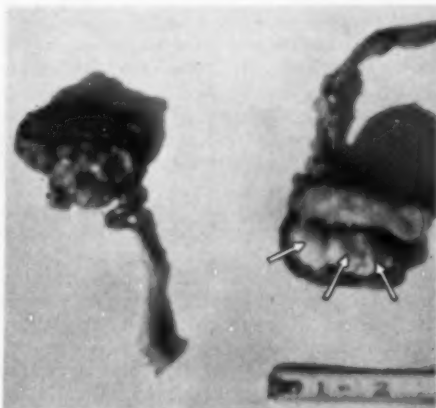


Fig. 2—Photograph of the scarred and shrunken kidneys of the dog showing calculi (arrows) in the pelvis of the right kidney.

nasia was performed. At necropsy, both kidneys were greatly shrunken and scarred; four calculi were found in the pelvis of the right kidney (fig. 2). The signs of illness

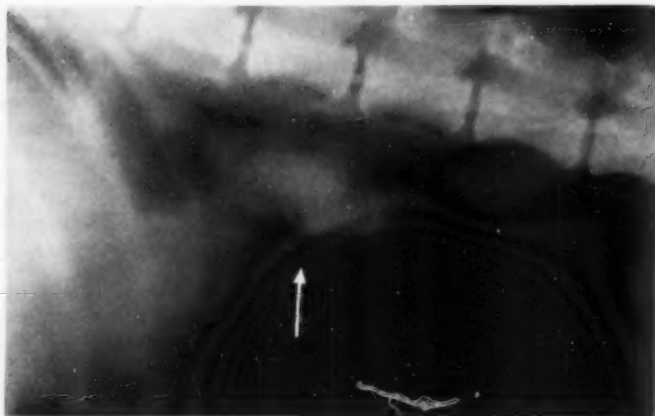


Fig. 1—Lateral radiograph of the abdomen of the dog showing an opaque shadow in the region of the kidney (arrow).

weight loss, intermittent vomiting, and polydipsia. The present signs were anorexia, anemia, depression, weakness, subnormal temperature, and ulceration of the buccal mucosa.

doubtless had been due to renal failure.—*Earl A. Grimm, D.V.M., Wayne D. Bradley, D.V.M., and Neal S. Nelson, B.S., members of the staff, Gordon Animal Hospital, Chicago, Ill.*

## What Is Your Diagnosis?

*History.*—A male Standard Poodle, 2 years old, had a slow-developing mass in the region of the right cervical lymph node. The enlargement was about 4 to 5 cm. in diameter, covered with skin and hair and, when palpated, it was tender

**Make your diagnosis from the picture below—then turn the page ►**



Figure 1

and movable. There was a question as to whether the other peripheral lymph nodes were enlarged. The mass was excised intact and the wound healed promptly.

The dog was returned four weeks later. The wound in the neck had opened and there was a nonpurulent exudate. Peripheral lymph nodes in all parts of the body were enlarged; some were as much as 2 cm. in diameter. Two toenails, the third on the left forefoot, and the third on the right hindfoot, had sloughed, leaving granulating wounds. In addition, there was a soft somewhat fluctuating swelling on the medial side of the right carpus. A lateral radiograph of the thorax showed no lesions. A dorsovolar radiograph of both forepaws was then taken. A photograph of this film is presented for diagnosis.

## Here Is the Diagnosis

(Continued from preceding page)

*Diagnosis.*—Systemic blastomycosis with bone lesions in a dog.\*

*Comment.*—Blastomyces were found in histological sections of the original lesions. They were missed when the sections were first examined, but a later search revealed a limited number of organisms in the lymph tissue adjacent to the inflammatory process; no organisms were found in the abscess or in the inflammatory tissue. Blastomyces were also found in aspirated material from the swelling at the carpus.



Fig. 2—Bone lesions of blastomycosis in (a) the third phalanx of the third digit of the right forefoot; and (b) the second and possibly other left metacarpal bones.

Our readers are invited to submit case histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

This case was submitted by Dr. Jack F. Marcus, Lake Forest, Ill.

\*This dog was from the suburban North Shore area of Chicago, where an occasional case of canine blastomycosis is found. The dog's kennel mate remained well.

## Response of Dogs to Distemper Vaccination—An Abstract

It is generally assumed that distemper virus (DV) is widely distributed and that contact may occur early in the dog's life; therefore, immunization must be considered at an early age. The object should be to replace natural immunization in which frank disease and deaths occur by an artificial method which produces lasting immunity without disease manifestations. This project was set up to determine factors affecting the serum-neutralizing antibody response, or resistance to challenge with virulent virus, of mature and young dogs and foxes following vaccination.

Serum-neutralizing antibody estimates, following vaccinating with egg-propagated virus, reached a ceiling in approximately one month and slowly declined for 18 or more months. Challenge of these vaccinated animals, when their serum antibody approached 0, revealed that they were refractory to intracerebral virulent virus exposure.

A controlled test has validated a previous suggestion that, under natural conditions, DV neutralizing antibody is maintained at high levels in urban dog populations by repeated contact with the virus. Limited data indicated that the antigenic stimulus of formalin-inactivated vaccine was sufficient to provide resistance. Inasmuch as the egg distemper virus replicates *in vivo*, presumably a more potent immunological attack may result than that provided by a fixed quantity of killed vaccine.

The immune status of the dam affects the immunizability of the young pup. High levels of passively transmitted maternal immunity may interfere with early vaccination, using a single dose of egg-adapted virus. Vaccination should be accomplished when the passive antibody conferred by the dam is at a level which will not interfere with active immunization. Pups from some immune dams could be immunized when vaccinated at 14 days of age; however, those from hyperimmunized dams could not be uniformly protected by vaccination at 42 days of age.

It may be hypothesized that pups from distemper-susceptible dams could be successfully immunized with egg-adapted virus at a few days of age; however, the practitioner should be warned that only

rarely would such bitches be encountered. Although it is hazardous to speculate, it is suggested that vaccination with egg-propagated virus should be accomplished when pups are 6 to 8 weeks old. It is still possible, in some instances, that there may be interference to this vaccine by passively transmitted antibody from the dam. Repetition of the vaccination, two weeks later, would tend to minimize this interfering effect.

In another trial, suckling pups, which had received maternal immunity by prenatal placental transmission and postnatally in the colostrum, responded to virulent exposure by the development of active immunity without manifestations of frank disease.

We can never expect complete success from any preventive measure. Dogs differ greatly in their susceptibility to infection and in the readiness with which they respond with antibody production. It is as foolish to condemn a method for a few failures to protect as to proclaim it successful because of a few instances of apparent protection.—[R. L. Ott, J. R. Gorham, and J. C. Gutierrez: *Distemper in Dogs. II. The Response to Vaccination*. *J. Am. Vet. Res. In press.*]

## Erysipelatous Arthritis in Rabbits

Arthritis was induced in 13 of 14 rabbits by repeated small intravenous injections of *Erysipelothrix rhusiopathiae*, and in 6 of 9 when given one injection. The developing arthritis, studied up to 850 days, showed a proliferation of granulation tissue and of the synovial membrane with a lymphocytic or plasma reaction. One rabbit, given a heavy single dose (21 billion organisms), developed severe systemic effects with the arthritis. Cortisone, given regularly, favored the induction of arthritis; however, the granulomatous reaction in the joints was less marked. Cortisone had no apparent effect when given six weeks after infection.—*Vet. Bull., Dec., 1956.*

Nine chickens given live *Leptospira canicola* and *Leptospira pomona* cultures, intravenously, showed no signs of infection but developed titers up to 1:128,000 which persisted for several months. Live cultures given intramuscularly produced no titers.—*Vet. Bull., Dec., 1956.*

## Bovine Mastitis

GUEST EDITORIAL (Requested)

The relation of economics and veterinary service has been properly emphasized in previous editorials. No greater opportunity awaits the veterinary profession than to prove how valuable professional services can be, through the use of a well-organized approach to the control of bovine mastitis.

Herds under well-planned control programs, in which the dairyman and the practicing veterinarian cooperate fully, have regularly produced more milk of higher quality. A reduction in the daily use of antibiotics as udder infusions usually follows. However, repeated veterinary examinations of the herd and equipment are necessary in order to make a control program effective. Surgical cleanliness and appropriate therapy are essential parts of the program.

One of the most frequent questions asked by a local veterinarian, when a paper has been given on bovine mastitis, is: "Can satisfactory mastitis control be obtained in large herds without laboratory aid?" It often can. Experience suggests that the application of a few rules of management would accomplish much in the average herd regardless of size. No one knows exactly which rules are most important, and authorities differ in the estimate of the value of each hygienic measure. It is also true that some herds are freed of *Streptococcus agalactiae* infection successfully, without much attention to management practices.

### HERD MANAGEMENT (HYGIENE)

The dairymen and practitioners who have followed a few basic rules required for the production of high quality milk believe that good herd management tends to reduce udder infection. Further application of these principles accomplishes much in the average herd regardless of size. Adherence to these rules is essential in the prevention of acute and chronic mastitis, whether or not laboratory aid is available to practitioners.

Among these principles, housing and milking methods which assure that neither gross nor less obvious teat injuries occur are most essential. Dipping the tip ends of the teats of each cow after each milking helps to inhibit bacterial growth in the

milk at the teat orifice and favors wound healing.

A general milking plan that requires that all first-calf heifers be housed in a separate barn and milked by different personnel and separate machines is invaluable, especially in larger herds. In small herds, the young mastitis-free cows should be placed at one end of the stable and milked first. After the herd is milked, the rubber parts of the milking machine must be thoroughly cleaned and ready for the normal young cows at the next milking.

The cows whose udders are definitely and obviously unsound are highly susceptible to acute or chronic mastitis. Many of these cows should be sold for beef. Some could be used for nurse cows or given a long dry period with repeated treatments.

Many additional details regarding the sanitation, mechanical efficiency, and use of milking machines should be understood by all the practitioners in dairy cattle areas. This might require special refresher courses dealing with the milking machine in relation to mastitis. The local practitioner is the most logical person to criticize or praise the dairyman for his methods. For example, he can instruct an owner in boiling the inflations in lye and resting them for a week, using two sets. Other people would be less able to impress the owner with such advice.

To insure good machine milking will require the combined ingenuity of the owner, the men who do the milking, and the practitioner responsible for the health of the herd.

The preparation of the cow for milking should include the use of a black Bakelite strip plate or strip pail before each milking. This is a fair indicator of the intensity of the mastitis problem in a herd.

Interpretation of the results of this test is an excellent means of diagnosis in the hands of an experienced veterinarian, but it leaves him wondering what infections are causing the abnormal milk that he discovered by this test.

The all too common practice, even in the ultramodern-appearing stables, of using a single plastic sponge or cloth in plain water or weak antiseptic solution to prepare a group of 10 to 50 cows for the milking process is a discredit to educated dairy-

men. The procedure of moving from cow to cow, either when stripping after the machine or when hand milking, should call for cleaning the hands in antiseptic solution between cows. Individual paper or sterile cloth towels are essential, particularly in cold climates, when washing teats and udders. Results may be better with no washing than where a poor job is done.

Owners expect and will pay for good results, and good results depend, in large part, on the skill and training of the man who milks the cows.

#### BREEDING FOR RESISTANCE

It is gratifying and encouraging to know that the personnel of animal husbandry departments, and of better breeding establishments, are advocating the development of cattle with compact udders and teats of normal length as one means of preventing mastitis caused by trauma. This, plus many other possible genetic factors which may predispose to mastitis, deserve study by veterinarians and others whose advice is used in artificial breeding circuits.

#### LABORATORY AIDS

There is also the question, "Should a complete laboratory diagnosis be made available for each dairy herd?" Certainly when an owner is ready to use the complete diagnosis, it is highly desirable from the standpoint of the practicing veterinarian to have such reports as an accurate guide in solving the problem in the herd. Procuring the samples and processing necessary reports on the herd entails labor and expense. Again, it should be emphasized that for best results the laboratory-guided program, coupled with the use of good management practices, would be better than either one alone.

What should we expect to learn from the results of the use of Murphy's CAMP test,<sup>1</sup> or from any other cultural examination of the milk from each cow in a herd? We should learn which quarters of the udders of each cow in herds under supervision are shedding *S. agalactiae*. This organism is recognized as one of the most important infections in chronic mastitis and is one udder pathogen that can be completely eliminated from a herd of any size.

<sup>1</sup>Murphy, James M., Stuart, Ortha M., Reed, F. I.: An Evaluation of the CAMP Test for the Identification of *Streptococcus Agalactiae* in Routine Mastitis Testing. *Cornell Vet.*, 42, (Jan., 1952): 133-147.

Furthermore, it can then be kept out more effectively, under most circumstances, than were the causative agents of brucellosis and tuberculosis when eradication of these diseases was started. Herds raised free from this particular streptococcal infection have been observed and, if housed and milked properly, will produce well and have a minimum of swollen or diseased quarters from any cause over long periods. Few large herds are maintained free of this infection, but many should and will be.

Do we know enough to control mastitis under all circumstances even with laboratory aid? The honest answer is "no," but let us hasten to state that full use of existing knowledge, if coupled with an accurate diagnosis, would come surprisingly close to doing this. Further research to determine the facts regarding other important causes of bovine mastitis and methods of their control should be liberally supported.

#### THE PRACTITIONER

What is the practitioner's role in this program? He should be the first to demonstrate to dairymen, to people conducting veterinary research, and to legislators the need for funds for laboratory aid in diagnosis and for further clinical and laboratory research. The practicing veterinarian should work closely with state-supported laboratory personnel in eradicating *S. agalactiae* infection from all dairy herds.

Once *S. agalactiae* has been eradicated from a herd, the education and experience required by those working in the dairy barn with the cows should result in less need for udder infusions, and the stage should be set for the much needed research on other pathogens that infect udders and destroy the usefulness of individual cows.

The major objectives in dealing with bovine mastitis, namely (1) a better quality of milk before pasteurization and (2) a product free of adulteration with antibiotics, should always be kept in mind. The latter can be achieved within six months after an effective program of prevention and specific treatment has been applied to a herd. The results of achieving these two things will also mean that the product will have greater consumer acceptance because of its higher quality, and the increased milk production in the herds will mean greater profits to the dairyman.—M. G. Fincher, D.V.M., Ithaca, N. Y.

# Current Literature

## ABSTRACTS

### Gastrointestinal Nematodes of Cattle

Keys are given which specifically provide for the identification of the third-stage larvae of eight species and the fourth-stage larvae of nine of 11 nematode species, *Trichostrongylus axei*, *Trichostrongylus colubriformis*, *Ostertagia ostertagi*, *Haemonchus contortus*, *Cooperia punctata*, *Cooperia pectinata*, *Cooperia oncophora*, *Nematodirus helvetianus*, *Strongyloides papillosus*, *Bunostomum phlebotomum*, and *Oesophagostomum radiatum* commonly found in the gastrointestinal tracts of cattle in the United States.—[Frank W. Donvres: *Keys to the Identification and Differentiation of the Immature Parasitic Stages of Gastrointestinal Nematodes of Cattle*. *Am. J. Vet. Res.*, 18, (Jan., 1957): 81-85.]

### Vibrio Fetus and Alterations of the Bovine Uterus

Intrauterine exposure of dairy heifers in estrus to *Vibrio fetus* resulted in the "repeat breeding" syndrome. Intravenous or intrauterine inoculation of pregnant heifers resulted in premature births, salpingitis, endometritis, and generalized inflammatory edema of the fetal membranes. The blood serum-agglutination test was unreliable in detecting vibriosis in the experimentally exposed as well as in field cases.—[J. Simon and S. H. McNutt: *Histopathological Alterations of the Bovine Uterus. I. Studies with Vibrio Fetus*. *Am. J. Vet. Res.*, 18, (Jan., 1957): 53-66.]

### Use of Piperazine on Ascarid-Infected Pigs

One-tenth therapeutic dose per day of polymeric piperazine-1-carbodiithioic acid given in the feed one week prior to, and three weeks after, infection failed to prevent migration of *Ascaris lumbricoides* larvae. Respiratory embarrassment and liver scarring resulted, although no worms were recovered from the small intestines of any of the pigs. Uninfected control pigs showed no liver involvement or respiratory embarrassment.—[William D. Linquist: *The Use of Low-Level Piperazine on Pigs Experimentally Infected with Ascaris Lumbricoides*. *Am. J. Vet. Res.*, 18, (Jan., 1957): 119-120.]

### Arthritis in Swine Due to Erysipelothrix Infection

A proliferative rheumatoid arthritis was produced experimentally in swine with cultures of *Erysipelothrix rhusiopathiae*. Since fibrotic renal lesions and degenerative adrenal glands were a common finding, the effects of exogenous adrenal steroids were studied in normal and arthritic animals.

Biweekly injections of 1 Gm. of desoxycortone acetate (DCA) resulted in paralysis and death in 2 normal 120-lb. swine. One shoat that received

500 mg. of DCA twice weekly developed polydipsia, polyuria, and recurring paralysis but survived. At necropsy, increased cerebrospinal fluid, kidney infarcts, and atrophic adrenals were present in all DCA-treated swine.

The animals receiving the high level of hormone showed increased synovial fluid, serosanguineous effusions in some joints, and some proliferation of the synovial villi. The addition of cortisone (75 mg. per day) failed to prevent the arthritic changes presumably produced by DCA overdosage.

In 6 of 7 shoats with experimentally produced erysipelas arthritis, DCA augmented the pathological course of the disease. Amelioration of the symptoms occurred in 4 swine with acute erysipelas arthritis when DCA was supplemented with cortisone. The relationship of adrenal and kidney pathology to the development of arthritis in swine is discussed.—[D. Sikes, G. M. Neber, and L. P. Doyle: *Studies on Arthritis in Swine. III. The Influence of Desoxycorticosterone in Normal and Arthritic Swine Due to Erysipelothrix Infection*. *Am. J. Vet. Res.*, 18, (Jan., 1957): 101-108.]

### Emulsion-Type Erysipelas Bacterin in Weanling Pigs

An emulsion-type erysipelas bacterin was prepared by processing 370 ml. of a formalin-killed, aluminum hydroxide-adsorbed *Erysipelothrix rhusiopathiae* culture, 590 ml. of light mineral oil (bayol F), and 40 ml. of manide mono-oleate (arlacel A).

Pigs of weaning age were vaccinated subcutaneously with 5 ml. of this emulsion bacterin. The immunity of groups of these swine was challenged by the percutaneous route at varying intervals after vaccination. The emulsion-type bacterin was shown to elicit an immunogenic response of longer duration than aqueous, formalin-killed, aluminum hydroxide-adsorbed bacterin. Pigs receiving the emulsion bacterin in this experiment developed a practical immunity lasting at least 237 days.—[N. K. Jungk and F. M. Murdock: *An Emulsion-Type Erysipelas Bacterin. I. Duration of Immunity in Pigs Vaccinated at Weaning*. *Am. J. Vet. Res.*, 18, (Jan., 1957): 121-125.]

### Effect of Antibiotics on Vibriosis

Seven antibiotics at different levels, alone and in combination, were added to semen diluted at a rate of 1:25 and 1:5 with skim milk diluent. Samples of diluted semen were stored at 5 C. Optimum levels for sperm motility were 500 µg. of dihydrostreptomycin, 250 µg. of streptomycin (500 µg. was satisfactory up to 2 days), 500 µg. of neomycin, 500 units of penicillin G, and 1,000 µg. of polymyxin B. Aureomycin® and terramycin® were highly toxic at levels of 250 µg. and 125 µg., respectively.

Dihydrostreptomycin and streptomycin were more effective against *Vibrio fetus* in the diluent than were neomycin, penicillin, polymyxin, aureo-

mycin, and terramycin. Dihydrostreptomycin (500 µg. level) was the most suitable single antibiotic used with respect to sperm toxicity and destruction of *V. fetus*. Antibiotic combinations employed were not spermicidal and produced a greater effect against the organism than either of the antibiotics acting alone, with one exception.—[L. R. DiLillo, Leo J. Poelma, and John E. Faber: *Effect of Antibiotics on Vibrio Fetus and Bovine Spermatozoa*. *Am. J. Vet. Res.*, 18, (Jan., 1957): 86-92.]

### Infectious Synovitis Control

Starting with 50 Gm. per ton of feed, the level of chlortetracycline, oxytetracycline, and an equal mixture of these two was increased by 25 Gm. increment up to 200 Gm. per ton, with a maximum of 250 Gm. per ton included. Streptomycin, penicillin, and equal parts of these two were fed at 100 and 200 Gm. per ton of feed. Streptomycin was also fed at 400 Gm. per ton. These medicated feeds were given continuously from 48 hours before inoculation to the end of the 18-day experimental period.

Ten 5-week-old chicks per pen were experimentally exposed by inoculation into the foot pads with 0.25 ml. of a yolk culture of the infectious synovitis agent.

Levels as low as 50 Gm. per ton of the tetracycline antibiotics showed some therapeutic value. There was a reduction in the number of birds showing swelling in the inoculated foot pad when chlortetracycline was used at 75 Gm. per ton and above, and no birds showed lesions when the drug was given at 200 Gm. per ton. The reduction in the number of birds showing swelling in the oxytetracycline and the combination of oxytetracycline and chlortetracycline group did not start until the 125-Gm. level was used. The latter drugs did not give complete control at any of the levels.

From this trial, it was concluded that the optimum level of chlortetracycline to control infectious synovitis in 5-week-old chicks was approximately 200 Gm. per ton of feed. With oxytetracycline or equal parts of chlortetracycline and oxytetracycline the levels needed to control synovitis would be somewhat higher. Streptomycin, penicillin, and equal parts of streptomycin and penicillin given in the feed did not alter the course of the infection.—[N. O. Olson, D. C. Shelton, J. K. Bletner, and C. E. Weakley, Jr.: *Infectious Synovitis Control. II. A Comparison of Levels of Antibiotics*. *Am. J. Vet. Res.*, 18, (Jan., 1957): 200-203.]

### Reservoir for Newcastle Disease Virus

A study of the hemagglutination (HA) test, using aqueous humor, was made with 995 groups of chickens submitted for routine disease diagnosis. It was found that approximately 83 per cent of the adults and 64 per cent of the growing birds were HA-positive, yet signs of frank Newcastle disease were rarely present. Correlation studies between HA reactions and various disease condi-

tions among chickens revealed a higher incidence of positive reactions in adult females with reproductive disorders than in birds not having this problem. In chickens less than 5 months old, there was a correlation between positive HA reactions and air sac infections. In a study of the relationship between HA-positive reactions and vaccination status, no appreciable difference could be detected among adults which had not received Newcastle disease vaccines and those which had been vaccinated during their growing age.

Chicken embryo inoculation studies resulted in first-passage isolation of the Newcastle disease virus from the aqueous humor of 111 of 127 HA-positive birds. All first-passage viruses isolated were found to be lethal to nonimmune test chicks. It was concluded that the aqueous humor is a reservoir for the Newcastle disease virus in a large percentage of chickens in southern California.—[D. S. Clark, E. E. Jones, and F. K. Ross: *Further Studies with the Aqueous Humor of Chickens as a Reservoir for the Virus of Newcastle Disease*. *Am. J. Vet. Res.*, 18, (Jan., 1957): 204-206.]

### Toxicity of Malathion in Chickens

Chickens dusted with 4 per cent malathion four times at weekly intervals did not develop signs of intoxication, nor did chickens, 4 weeks old, given 100 mg. per kilogram of body weight of malathion orally twice within seven days. Chickens, 4 weeks old, receiving 200, 400, and 600 mg./kg. of the drug showed increased signs of intoxication as the dose was increased. The L.D.<sub>50</sub> for 3-week-old birds apparently is between 200 and 400 mg./kg. Younger birds appeared to be more tolerant to its effects at the 200 mg./kg. level than did older birds (1 yr. old). The symptoms of malathion poisoning in chickens are described.—[S. M. Gaafar and R. D. Turk: *The Toxicity of Malathion in Chickens*. *Am. J. Vet. Res.*, 18, (Jan., 1957): 180-182.]

### Histopathology of Leucocytozoon Simondi Infection

Histopathological changes of a systemic nature in ducks with severe, fatal *Leucocytozoon simondi* infection were liver necrosis, periportal and diffuse lymphocytic infiltrations in the liver, hemosiderosis in the liver and spleen, splenomegaly from congestion and macrophage proliferation, and hyalinization of the splenic adenoid sheaths. Ducks with moderately severe infection showed similar but less severe alterations, and those with light, nonfatal infection showed none. Local host tissue reaction occurred only in relation to megaloschizonts in the brain and lung tissue, not in other organs nor in relation to hepatic schizonts or gametocytes. Megaloschizonts were enveloped by a capsular wall of reticular fiber which, in all organs except the brain, developed from condensation of reticular fibers on the surface of the expanding

megaloschizonts. Exoerythrocytic stages and the essential morphological differences between megaloschizonts occurring in the different organs are noted.—[James W. Newberne: *Studies on the Histopathology of Leucocytozoon Simondi Infection*. *Am. J. Vet. Res.*, 18, (Jan., 1957): 191-199.]

#### Polyvalent Toxoid for Botulism

A method is described for the preparation of toxoids of *Clostridium botulinum*, types A, B, and C. The entire procedure from incubation of culture to removal of formalin from the toxoid may take place in a closed system. One injection of a mixture of alum-precipitated toxoids thus produced has been found efficacious in protecting mink from large doses of type C toxin, the most common cause of epizootics on mink farms.—[J. F. Bell, R. K. Farrell, and J. R. Gorbam: *A Polyvalent Toxoid for Botulism*. *Am. J. Vet. Res.*, 18, (Jan., 1957): 167-170.]

### FOREIGN ABSTRACTS

#### New Findings in Bovine Ketosis

The author reports excellent results in the treatment of 52 cases of ketosis with dihydrotachysterol (A.T. 10) with or without calcium gluconate. He states that, in ketosis, the serum concentration of phosphorus is usually higher than normal, while the serum-calcium level is always lower. It is theorized that the comatose form of ketosis is actually a manifestation of hypocalcemia rather than intoxication from the blood ketones. Thus, calcium therapy is always indicated in the treatment of ketosis as well as in parturient paresis.

The author proposes this sequence of events leading to the development of ketosis: a primary hypofunction of the parathyroids, leading to lowered serum-calcium, along with an impaired function of the anterior pituitary with decreased adrenocorticotropin (ACTH); there is hypofunction of the adrenocortices, with the consequent disturbance in the metabolism of lipids.

According to the author, the use of ACTH or the cortisones constitute symptomatic therapy similar to the use of glucose, acethylmethionine, or sodium propionate.—[H. Bomer: *L'acétonémie des Vaches laitières traduit-elle un hypofonctionnement des parathyroïdes?* *Rec. méd. vét.*, 132, (1956): 872-873.]—R. F. VIGUE.

### BOOKS AND REPORTS

#### Antibiotics Annual

More than 150 papers presented at the third annual Symposium on Antibiotics, held in November, 1955, in Washington, D.C., are published in this edition of "Antibiotics Annual." Of these, 30 are on new antibiotics and 20 are from 15 foreign countries.

Some of the subjects of special interest to veterinarians are: antibiotic therapy for burns; antibiotics and the bone marrow of well-nourished

and undernourished dogs; cycloserine for stubborn urinary infections; antibiotic and vitamin combinations; vitamin-fortified antibiotics in acute radiation syndrome of dogs; antibiotics and growth response in chicks; oxytetracycline for synovitis of chickens; antibiotic preservation of meats; mastitis; and chlortetracycline for pigs with atrophic rhinitis. The role of ACTH and cortisone in tuberculosis therapy is also discussed.

Most veterinarians, but especially those in teaching and research, will find this book of value.—[*Antibiotics Annual, 1955-1956*. Edited by Henry Welch and Felix Marti-Ibanez. Illustrated. 994 pages. Medical Encyclopedia, Inc., New York, N. Y. 1956. Price not given.]

#### Animal Diseases in South Africa

The third edition of this book is enlarged and includes a description of a number of diseases not reported in the previous edition, including the new Wesselsbron virus. This is now a large reference book and concerns itself only with infectious diseases of domestic animals. Most of these are peculiar to Africa and, on the whole, are inadequately dealt with in other texts. Diseases like bovine pleuropneumonia, botulism, anaplasmosis, the piroplasmoses, theilerioses, trypanosomiasis, and the many important virus diseases of Africa are some of those discussed in great detail, but the book also covers many other important diseases of worldwide importance such as mastitis, tuberculosis, and leptospirosis.

The book is carefully written and fully illustrated. Special features are the excellent historical accounts of each disease and the exhaustive review of the literature with a thorough bibliography.

This excellent book should be available in every veterinary library and research laboratory and in the office of every state and federal regulatory officer to supplement the 1954 U.S. Livestock Sanitary Association handbook "Foreign Animal Diseases."—[*Animal Diseases in South Africa*, By M. W. Henning. 1239 pages. 3rd ed. (completely revised). Central News Agency Ltd., Johannesburg, South Africa. 1956. Price 5 pounds, 5 shillings (approx. \$15).]—V. R. KASCHULA.

#### Principles and Methods of Sterilization

After a brief history on the development of sterilization, the author discusses the thermal destruction of microorganisms, principles of steam sterilization, minimum standards for sterilization, modern surgical supply sterilizer, preparation and sterilization of dressings and surgical instruments, and gaseous and chemical disinfection. The care of the instruments and equipment used during the process of sterilization is also considered. In the veterinary field, this book would be helpful to those working in research laboratories and to hospital owners.—[*Principles and Methods of Sterilization*, By John J. Perkins. 340 pages. Illustrated. Charles C Thomas, Springfield, Ill. 1956. Price \$8.00.]

## Fellowship Offered in Biophysics

The University of Kansas announces an opening for a graduate veterinarian interested in obtaining a grant to work on a master's degree in radiation biophysics.

The grant for the fellowship was given by the Mark L. Morris Animal Foundation for the study of the role of the thyroid and liver in canine obesity.

Qualifications for the grant include an interest in research with a background in mathematics, chemistry, and biology. Interested veterinarians should write Dr. Frank E. Hoecker, Radiation Biophysics Program, University of Kansas, Lawrence, Kan.

## A.M.A. Rural Health Conference

The twelfth national conference on rural health sponsored by the American Medical Association will be held March 7-9, 1957, at the Brown Hotel, Louisville, Ky.

Among the presentations planned for this meeting are description of modern day medical care, the impact of modern living, and rural economics in relation to health care; the migrant labor problem. Discussion periods are being planned with the entire group participating.

## Dr. Catcott Joins Public Health Service

Dr. Earl J. Catcott, a former professor of veterinary medicine at Ohio State University, has been commissioned in the U.S. Public Health Service. He is currently assigned to the California State Health Department and is stationed at Los Angeles. Dr. Catcott has joined a group of investigators who are studying the effects of smog on human and animal health.

## Inter-American Veterinary Medical Symposium Held

An Inter-American Veterinary Medical Symposium, sponsored by the Asociación Médico Veterinaria de Puerto Rico, was held at the Caribe Hilton Hotel on Nov. 30-Dec. 2, 1956.

The scientific program included the following speakers and subjects: Dr. Jack O. Knowles, Miami, Fla. (management of distemper); Dr. Raúl Oms Narbona, La Habana, Cuba (some aspects of poultry breeding); Dr. S. J. Roberts, Ithaca, N. Y. (dairy problems); Dr. Robert L. Burkhart (aureomycin therapy of major domestic animal diseases); Dr. W. W. Armistead, president-elect of the AVMA, College Station, Texas (newer surgical techniques); Dr. E. A. Benbrook, Ames, Iowa (the past, present, and probable future of the control of parasites in livestock); Dr. Manuel Ramirez-Valenzuela, México, D. F. (bovine rabies); and Dr. Frank Kral, Philadelphia, Pa. (bacte-

rial and fungus infections of the skin, including the ear, transmissible from animals to man). Moderators at the scientific sessions were Drs. Angel C. Pou, Ponce, Puerto Rico, and Adolfo A. Alvarez, Mayaguez, Puerto Rico.

In the matter of entertainment provided for veterinarians and their wives, an informal get together was held at the Isla Verde Airport on Friday, November 30, in the evening. On Saturday, a luncheon was held at the Borinquen Room of the Caribe Hilton Hotel at which those attending were greeted by Dr. A. Garcia Bird, president of the Puerto Rico Veterinary Medical Association, and addressed by Dr. Jaime Bagué of San Juan, Puerto Rico. A fashion show for the women was provided in the afternoon and a reception in the evening at the Borinquen Room.

On Sunday, December 2, a picnic at the beautiful Luquillo Beach was held. Many of the guests had the opportunity of driving a few miles up the mountains to view the only existing example of a rain forest in this hemisphere.

An interesting note at the scientific session was the use of an IBM Wireless Translator System providing presentation of lectures simultaneously in Spanish and English. Veterinarians from Cuba, México, Trinidad, Virgin Islands, Guatemala, Peru, and the United States attended the symposium.

s/O. A. LOPEZ-PACHECO, *Resident Secretary.*

## Veterinary Faculty Changes at Missouri

The following changes in personnel have been made at the School of Veterinary Medicine, University of Missouri, Columbia: Emmett L. McCune (MO '56), instructor, Department of Veterinary Bacteriology and Parasitology; and Harold B. Wright (MO '55), instructor, Department of Veterinary Pathology.

## Dr. Greene Joins University of Delaware Faculty

Dr. L. M. Greene has joined the staff of the Department of Animal and Poultry Industry, University of Delaware, effective Dec. 1, 1956.

Dr. Greene obtained his B.S. and M.S. degrees in poultry husbandry at North Carolina State College, and is a graduate of Michigan State University where he received his D.V.M. degree in 1939.

He was a member of the Veterinary Corps during World War II and, since 1947, has been poultry pathologist with the Veterinary Division, North Carolina Department of Agriculture, Raleigh.

Dr. Green will be stationed at the University of Delaware substation at Georgetown, Del., where he will conduct poultry disease research and poultry diagnostic work.

## AMONG THE STATES AND PROVINCES

### California

**Medical Research Association Meeting.**—The eighth annual meeting and dinner of the Medical Research Association of California was held on Thursday, January 17, in Los Angeles. Dr. Karl F. Meyer spoke on "Medical Research in the USSR." Dr. Meyer recently visited Russia at the request of the U. S. State Department. His report covered his experiences and observations on Russian research and education in biology and medicine. A number of members of the Southern California V.M.A. attended the meeting.

### Illinois

**Short Course on Poultry Diseases.**—A poultry disease short course for veterinarians was held at the College of Veterinary Medicine, University of Illinois, Urbana, Feb. 5-6, 1957. Enrollment was limited to 25 veterinarians and a fee of \$15 was charged.

Subjects included in the program were a slide review of poultry diseases and management practices; poultry public health problems; developing a poultry practice; bacteriological procedures (mediums, equipment, isolation and identification of common pathogens, antibiotic sensitivity tests); parasitological examination; new and old diseases; therapy and prophylaxis; poultry practice techniques (caponizing, de-beaking, handling of birds, blood collection, necropsy); virology and serology; pet bird management and disease problems; and a review of bacteriological, serological, necropsy, and diagnostic procedures.

### Indiana

**Tenth District Association.**—The following officers were elected for 1957 at the December, 1956, meeting in New Castle: president, W. E. Sharp, Union City; vice-president, R. H. Goodale, Muncie; and secretary-treasurer, J. S. Baker, Pendleton.

S/B. LA SALLE, *Publicity Officer.*

### Louisiana

**Conference for Veterinarians.**—The annual conference for veterinarians was held at Louisiana State University, Baton Rouge, Jan. 29-30, 1957. Those attending the conference participated in the dedication ceremonies of the new Veterinary Medical Research Center. President Wayne O. Kester represented the AVMA at the conference and the dedication.

### Maine

**State Association.**—The winter meeting of the Maine V.M.A. was held Jan. 16, 1957, at Pittsfield. A panel discussion on a series of

selected cases was a highlight of the meeting. Participating on the panel were Drs. Deane M. Chamberlain; Owen R. Stevens, Jr.; Myles J. Edwards; E. J. P. McDonald; Lewis B. Denton; S. W. Stiles; John M. Woodcock; and McClure Day.

The date of this meeting was established to permit several members to attend the conferences held at Cornell University and the University of Pennsylvania early in January. Reports were made by those who attended the conferences.

Officers elected for the following year are Drs. James Elliott, president; P. R. Brown, vice-president; and J. F. Witter, secretary-treasurer.

### Michigan

**Conference for Veterinarians.**—The thirty-fourth annual postgraduate conference, Michigan State University, was held Jan. 22-23, 1957. The entire morning program of the first day (Tuesday) was devoted to closed-circuit television demonstrations. A general session was held Tuesday afternoon, and the Wednesday program was divided into large and small animal sections.

The first official alumni reunion of the College of Veterinary Medicine, M.S.U., was held Tuesday evening, January 22.

### Minnesota

**State Association.**—The sixtieth annual meeting of the Minnesota State V.M.A. was held Feb. 4-6, 1957, at Minneapolis.

Out-of-state speakers included Drs. D. F. Bunce, Chicago, Ill.; John B. Herrick, Ames, Iowa; Elroy C. Jensen, Ames, Iowa; A. C. Jerstad, Puyallup, Wash.; Lloyd D. Jones, Omaha, Neb.; Wayne O. Kester (AVMA president), Washington, D. C.; L. C. Moss, Fort Collins, Colo.; W. B. Pritchard, Lafayette, Ind.; J. C. Siegrist, Bloomfield, N. J.; H. C. Smith, Sioux City, Iowa; and E. A. Woelffer, Oconomowoc, Wis.

Additional speakers appearing on the program were Mr. C. F. Chappel, Eli Lilly Co., Indianapolis, Ind. (new trends in animal feeding); and Eugene P. Cronkite, M.D., head, Division of Experimental Pathology, Medical Department, Brookhaven National Laboratory, Upton, N. Y. (fall-out, civil defense, and effects of radiation on man).

Mrs. E. A. Woelffer, first vice-president of the Women's Auxiliary to the AVMA, was the guest of honor at tea, and entertainment was sponsored by the Auxiliary to the Minnesota V.M.A.

### New Jersey

**State Association.**—The New Jersey V.M.A. sponsored a meeting of veterinarians and farmers during Farmers' Week, Jan. 24, 1957, in Trenton.

Veterinarians appearing on the program and their subjects were C. A. Manthei, cattle diseases; C. N. Dale, swine diseases; O. L. Os-teen, poultry diseases; and J. Whitlock, sheep diseases.

#### New York

**New York City Association.**—The regular meeting of the Veterinary Medical Association of New York City, Inc., was held on the evening of Wednesday, Jan. 9, 1957, at the New York Academy of Sciences, New York City. The guest speakers on the program were Dr. F. G. Fielder, Schering Corporation, Bloomfield, N. J., and Dr. W. C. Glenney, Ardmore, Pa.

s/C. E. DeCAMP, Secretary.

• • •

**State Board Examinations.**—The Board of Examiners, during the recent conference for veterinarians, held at the New York Veterinary College at Ithaca, set as the dates for the practical examination, June 12-13, 1957. This examination will be held at Ithaca.

The written examination will be held the week of July 8 at the following centers: New York, Albany, Syracuse, Buffalo, and Roches-

ter. Requests for applications and further information should be addressed to Mr. James O. Hoyle, secretary, State Board of Veterinary Examiners, 23 S. Pearl St., Albany, N.Y.

#### North Carolina

**State Board Examinations.**—The North Carolina State Veterinary Examining Board will hold a meeting on June 24-26, 1957, at the Grove Park Inn, Asheville, N. Car. Requests for further information should be addressed to James I. Cornwell, secretary, North Carolina State Veterinary Examining Board, 65 Beverly Rd., Beverly Hills, Asheville, N. Car.

#### Pennsylvania

**Dr. Korns Elected to State Legislature.**—Dr. William R. Korns, Somerset, Pa., was elected to the House of Representatives, Pennsylvania State Legislature, during the recent election.

### U. S. GOVERNMENT

**Veterinary Personnel Changes.**—The following changes in the force of veterinarians



### Closed Circuit Television at Michigan State University



At Michigan State University, in the fall of 1956, the College of Veterinary Medicine initiated the use of closed-circuit television in regular courses in surgical anatomy two hours per week and in small animal surgery one hour per week. The general arrangement is shown, with the surgery demonstrations (monitor), cameras, and three viewing screens functioning in the classroom before 120 students (senior and junior classes).

This method makes it possible to demonstrate a maximum of material to a large group in a limited time with a minimum of preparation. This is particularly true in demonstrating cadaver material (right inset) as well as surgical procedures (left inset).

Eventually, it should be possible to present relatively unobstructed views of detailed procedures which, like "Cinerama," will excel the views of anyone except the surgeon.

in the U.S.D.A. are reported as of Jan. 16, 1957.

#### TRANSFERS

Alexander H. Abramson, from Lakewood, N. J., to Camden, N.J.

George A. Ahrens, from Menominee, Mich., to Eau Claire, Wis.

George E. Aidman, from Rapid City, S. Dak., to Indianapolis, Ind.

Charles Barnes, from Ottumwa, Iowa, to Salt Lake City, Utah.

William H. Bassett, from Allentown, Pa., to Ottumwa, Iowa.

Walter W. Campbell, from Siloam Springs, Ark., to Rogers, Ark.

William W. Canon, from Chicago, Ill., to DeQueen, Ark.

Raymond Cheezig, from Worthington, Minn., to Omaha, Neb.

Donald M. Chesen, from Farmingdale, N.J., to Camden, N. J.

Jack E. Gross, from Oklahoma City, Okla., to Fort Smith, Ark.

Harold R. Henthorn, from Central City, Neb., to Worthington, Minn.

Wayne Hornaday, from Harrisonburg, Va., to Lakewood, N. J.

Walford J. Johnson, from Salt Lake City, Utah, to Sioux City, Iowa.

Robert D. McElwain, from Modesto, Calif., to Nephil, Utah.

Joseph O. Minnich, from Kansas City, Kan., to Menominee, Mich.

M. I. Ostrowski, from Chicago, Ill., to Union, Mo.

Thomas O. Roby, from Washington, D. C. to Beltsville, Md.

E. E. Saulmon, from Baton Rouge, La., to Washington, D. C.

James F. Sullivan, from Washington, D. C., to Beltsville, Md.

Frank R. Thorndike, from Sioux City, Iowa, to San Francisco, Calif.

Wladimir Wacyk, from Stanley, Va., to Broadway, Va.

L. E. Williamson, from New Market, Va., to Harrisonburg, Va.

Alexander M. York, from Modesto, Calif., to San Francisco, Calif.

#### RETIREMENTS

Maurice C. Duff, Frankfort, Ky.

William F. Fisher, Reno, Nev.

Walter J. Hall, Beltsville, Md.

Eugene Hyland, Riverside, Calif.

Howard D. Osborne, West Fargo, N. Dak.

Thomas E. Sanders, Rogers, Ark.

Pierre P. Soullard, Salem, Ore.

Roswell A. Telford, Watertown, S. Dak.

DeLois G. Tepfer, Fort Dodge, Iowa.

### STATE BOARD EXAMINATIONS

**NEW YORK**—June 12-13, 1957. Practical examination, Ithaca. Mr. James O. Hoyle, secretary, 23 S. Pearl St., Albany. Week of July 8, 1957. Written examinations: New York City, Albany, Syracuse, Buffalo, Rochester.

**NORTH CAROLINA**—June 24-26, 1957. Asheville. Dr. James I. Cornwell, secretary, 65 Beverly Road, Beverly Hills, Asheville.

**TENNESSEE**—June 24-25, 1957. Nashville. Dr. W. O. Greene, secretary, State Office Bldg., Nashville.

### DEATHS

Star indicates member of AVMA

★**John F. Chipman** (KCV '14), 63, died in Orlando, Fla., on Dec. 9, 1956, after several months' illness. Dr. Chipman had served for a number of years in the tick eradication program of the BAI, U.S.D.A., in Georgia and Florida and in the Meat Inspection Division in Chicago, Ill., and Louisville, Ky. He retired from the Meat Inspection Service in Louisville in 1952 and at the time of his death he was serving as a veterinary supervisor of the State Meat Inspection program of the Florida Livestock Board with headquarters in Orlando.

Dr. Chipman was a member of the Methodist Church and the Masonic Lodge in Louisville.

**F. F. Eckert** (CVC '02), 80, died Nov. 15, 1956, at Lancaster, Wis. Dr. Eckert practiced at Lancaster from 1903 until his retirement in 1943. Surviving are a sister and brother.

★**P. W. Flickinger** (MCK '06), 74, Greenfield, Iowa, died Nov. 17, 1956, as a result of injuries suffered in an automobile accident. Mrs. Flickinger was fatally injured in the same accident, her death occurring Jan. 5, 1957. Dr. Flickinger was a member of the AVMA.

★**Glenn I. Luymes** (ISC '50), 33, Galva, Ill., was killed in an automobile accident Dec. 20, 1956. Dr. Luymes joined the AVMA in 1950.

**Charles E. Morgan** (ONT '17), 63, Toronto, Ont., died while on a hunting trip Nov. 17, 1956. Dr. Morgan was on the staff of the Health of Animals Division, Canada Department of Agriculture. His widow and two sons survive.

**Maurice A. Quinn** (NYS '16), 64, Norwich, N. Y., was killed in an automobile accident Oct. 10, 1956. Dr. Quinn had operated a commission auction market and was superintendent of races at the Chenango County (N. Y.) Fair for more than 30 years.

**Hubert F. Ronnenberg** (IND '15), 65, Houston, Minn., died Nov. 13, 1956. Dr. Ronnenberg had practiced in the Houston area for 32 years and was a highly respected member of this community. He was a member of the Minnesota State V.M.A. He is survived by his widow and a daughter.

★**Clifford C. Sockman** (OSU '06), 79, Deshler, Ohio, died Nov. 10, 1956, of pneumonia. Dr. Sockman had practiced in Deshler for 50 years. He was a member of the AVMA for 40 years. Surviving are the widow, two sons, and two daughters.

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### Questions and Answers on Social Security

**Question**—Dr. White, a self-employed veterinarian, was 62 years old in November, 1956. His net earnings for the year 1956 (the first year of coverage for self-employed veterinarians) was \$4,200. He filed his income tax return in January, 1957, for the year 1956 and paid a self-employment tax of \$126 (3% of net earnings of \$4,200). He then became ill for one and a half years and had no earnings until he resumed his practice in July, 1958. His net earnings for the year 1958 (annual earnings are used for the self-employed) amounted to \$2,100. He continued his practice until his retirement age 65 in November, 1959. His net earnings in 1959 were \$4,200. What will his benefits be under the Social Security Act if he were single and also what will they be if he were married?

**Answer**—In order to be eligible for Social Security benefits, one must have reached retirement age (65 for men and 62 for women) and have been in work covered by the Social Security law for a certain length of time. The length of time that one must work in covered employment in order to acquire an insured status depends on the individual's age. The amount of work required is measured in "quarters of coverage." The self-employed get four quarters of coverage for the year if they have net earnings of \$400 or more in a year.

One can become "fully insured" if he has one quarter of coverage for each two full calendar quarters after 1950 and up until the time he reaches retirement age. At least six quarters of coverage are necessary in every case, and in no case does one ever require more than 40 quarters of coverage. There is also a special provision for a fully insured status. This special rule provides that an individual will be fully insured if he has quarters of coverage in all but four of the calendar quarters after 1954.

In Dr. White's case, he requires 17 quarters of coverage under the normal rule or 15 under the special provision. Since, because of his illness, more than four of the calendar quarters after 1954 are not quarters of coverage, in his case, the special provision would not apply. Therefore, in order for Dr. White to qualify for benefits, he must have 17 quarters of coverage. Dr. White acquired four quarters of coverage for 1956, none for 1957, four for 1958, and four for 1959. He requires five more quarters of coverage in order to be eligible for Social Security benefits. He may acquire these additional quarters of coverage even after age 65. Accordingly, Dr. White may qualify for benefits if he continues his practice until 1961, and has net earnings of at least \$400 in 1960 and 1961.

**Question**—Dr. Gray, a self-employed veterinarian, became 65 years of age in February, 1956, and will report net earnings of \$4,200 from self-

employment for the year 1956. He will pay a self-employment tax of \$126 (3% of \$4,200) for the year 1956. During the last quarter of 1956, Dr. Gray sold his hospital and practice, and expects to work for his successor as an employee at \$350 per month during 1957. How will Dr. Gray report his 1957 earnings? Will he be eligible for retirement July 1, 1957, and what will his monthly benefit be?

**Answer**—During the year 1957, Dr. Gray's successor will report Dr. Gray's wages each quarter to the district director of Internal Revenue. Dr. Gray's employer should deduct 2¼ per cent each pay day from the gross wages paid to Dr. Gray during the year 1957, up to a maximum of \$4,200. Dr. Gray's employer will match Dr. Gray's contribution when he files the quarterly tax returns. If Dr. Gray terminates his employment at the end of June, 1957, after having been paid wages of \$350 per month, he can qualify for the maximum benefit of \$108.50 per month beginning July, 1957; however, if Dr. Gray continues to work through December 1957 and earns \$350 per month through the end of 1957, his benefit would begin in January, 1958, at the rate of \$108.50. If Dr. Gray works on a part-time basis after that, and doesn't earn more than \$1,200 a year, he may still receive his benefits for each month of the year. After he reaches 72 years of age, he would receive his benefits no matter how much he earns from then on.

#### Business Proceedings for 1956

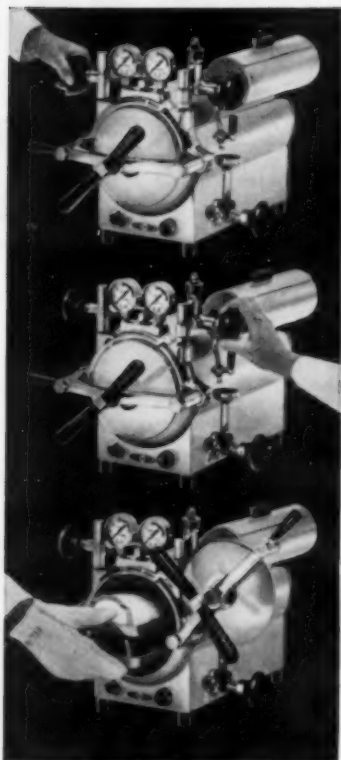
The Proceedings of the sessions of the House of Representatives, held on Oct. 13, 1956, preceding the Ninety-Third Annual Meeting, in San Antonio, are published as Part 2 of this issue (March 1, 1957) of the JOURNAL, and are mailed in the same envelope with Part 1 of this issue.

#### AVMA Research Fellowships Available

The Research Council of the American Veterinary Medical Association announces the availability of a number of fellowships for postgraduate training for the academic year, 1957-1958.

Any qualified person interested in graduate training may obtain application blanks and other information by writing to Dr. C. H. Cunningham, secretary, AVMA Research Council, Department of Microbiology and Public Health, College of Veterinary Medicine, Michigan State University, East Lansing, Mich.

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## investigators report

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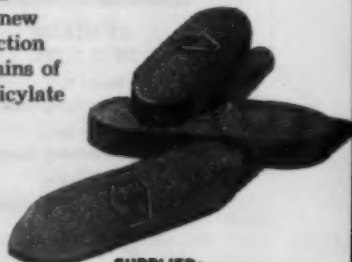
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Investigators state: "Even calves with very bloody feces and moribund aspect when first seen made . . . a surprisingly rapid and complete recovery."<sup>2</sup> Beneficial effects are often obvious in the first 12 hours of treatment, with complete recovery after only 3 or 4 doses.<sup>2</sup> Calves raised on milk-substitute show a recovery rate on ENTEFUR comparable to that of nursing calves. No toxic effects have been demonstrated during administration of higher than therapeutic doses for prolonged periods.<sup>2</sup> Bacterial resistance does not tend to develop during treatment with the *nitrofurans* and has not been demonstrated in vitro or in vivo with ENTEFUR.

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**DOSAGE:** 1 ENTEFUR Bolus Veterinary, small, for each 150 lbs. body weight, or less, twice daily for 2 or 3 days. In most cases, 4 doses are sufficient for full recovery.

**REFERENCES:** 1. Segard, C. P., *Farmer's Digest*, May, 1955.  
2. Henry, E. T., and Blackburn, E. G., *Vet. Med.*, in press.  
3. Bull, W. S., *N. Amer. Vet.*, in press.



**SUPPLIED:** ENTEFUR Boluses Veterinary, small (3 Gm. each) are available in box containing 6 envelopes of 4 boluses each.

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**93-95% recoveries**



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## Principles of Ethics

During the past several years, the Committee on Ethics has considered a number of inquiries concerning interpretations and applications of the Principles of Veterinary Medical Ethics of the American Veterinary Medical Association.

A series of discussions relating to various sections of the Principles will appear in this space from time to time. The first of the series appeared in the February 1, 1957, issue (adv. p. 34).

### Ethical Directory Listings

One section of the Principles of Veterinary Medical Ethics of the AVMA deals with directory listings. Four paragraphs are devoted to this subject.

*Paragraph 10.*—Advertising in a city, commercial, telephone, or any widely circulated directory is a violation of this code.

*Paragraph 11.*—A member who permits his name to be listed in directories in bold face type or who advertises his name or hospital or institution in any way differing from the standard style, type, or size used in the directory for the listing of professional groups (physicians, dentists, lawyers, nurses) is subject to the charge of unprofessional conduct.

*Paragraph 12.*—It is also unethical for a veterinarian to allow his name to be printed in public directories as a specialist in the treatment of any disease or in the performance of any service within the scope of veterinary practice.

*Paragraph 13.*—In principle, this section of the code of ethics is intended to improve the listing of names in such a way as to give all of them identical visual prominence.

One of the more common sources of complaint, locally at least, is the failure of some veterinarians to observe the rules for ethical listing in the classified sections of telephone directories or in "Red Book" directories.

Violations of ethical standards for directory listings can be charged to several causes: ignorance of ethical requirements; thoughtlessness or carelessness about this aspect of professional behavior; yielding to pressure of misguided directory space salesmen who do not realize, apparently, that veterinarians have the same rules of conduct as other ethical professional groups; and deliberate disregard of ethical standards. In years past, some new and inexperienced graduates made the mistake of employing boldface or display types of listings until they learned that such entries are unethical. In recent years, it is unlikely that any graduates from the accredited veterinary colleges are not well

informed on such matters, because practically all of them have been exposed to lectures and training on professional matters, including the important subject of ethics.

Much of the progress in recent years in eliminating unethical listings of veterinarians and other professional people from telephone directories has been made through the efforts of the local professional associations. Through frank and friendly discussion of the basic principles involved, it is usually possible to agree upon a course of action that will bring a high degree of cooperation and compliance among the practitioners in any given locality. The fact remains, however, that in some cities and areas, little has been done to "clean up" directory listings of veterinarians. Bad examples are easy to find, ranging all the way from bold face listings to box and display insertions of one inch to a quarter page, and even a half page.

It is hoped that the listing of sample ethical directory listings below will assist local groups in making necessary corrections:

#### *Ethical Directory Listings*

##### VETERINARIANS

Arcade Animal Hospital 16789 Grand Rd., J. J. Stiles, D.V.M.	2-6134
Brown, J. D., 116 Milltown Ave. (Practice limited to horses)	2-5647
Callendar, John J., 146 Elgin Ave. (House calls only)	3-5638
Dardis Pet Hospital, 12 "E" St., Dardis, Harry J., 128 Jackson (Res.) If no answer call	3-2465 4-0930 4-8970
Frank Small Animal Hospital 2828 Jennison St., George Frank, D.V.M. Albert Moore, D.V.M. Ofc. hours 1 to 5 p.m. & 6 to 8 p.m.	6-4576
Frank, George A., 1177 Jerico	5-6745
Kalbeck, E. E., 1289 Summit Ave. (By appointment only)	4-5324

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V-MC-J-117

## ORGANIZATION SECTION

Moore, Albert, 238 Palm Blvd. (Res.)	4-3687
If no answer call	6-4576
Mosier, Albert S.	5-3498
(Practice limited to cats)	
Stiles, J. J., 3620 Arcade Ave.	3-8967
Stokes Animal Hospital	
23489 Long John Rd.	2-4678
Farm service (John Wysong, D.V.M.)	2-5679
Ofc. hours. 10-12 a.m. & 2-5 p.m.	
Sundays and hol., 9-11 a.m.	
Stokes, John M., 1622 Elm St.	3-4765
Wysong, John, 3920 Jackson Ave.	3-9754

AVMA Committee on Ethics

WILTFONG, PHILLIP D.  
1419 O St., Aurora, Neb.  
D.V.M., Kansas State College, 1952.

### Applicants—Not Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., notice of all applications from applicants residing outside of the jurisdictional limits of the constituent associations, and members of the Armed Forces, shall be published in the JOURNAL for two successive months. The first notice shall give the applicant's full name, school, and year of graduation, post office address, and the names of his endorsers.

### First Listing

FORD, DOUGLAS T.  
Maple Cattle Breeders Association, Maple, Ont.  
D.V.M., Ontario Veterinary College, 1955.  
Vouchers: J. A. Henderson and F. J. Milne.

FOSTER, JAMES W.  
1442 — 14th Ave. N., Fort Dodge, Iowa.  
D.V.M., Iowa State College, 1954.  
Vouchers: O. Emerson and R. C. McCord.

ROGERS, OWEN L.  
217 Magruder, Mineral Wells, Texas.  
D.V.M., Texas A. & M. College, 1946.  
Vouchers: J. F. Melton and E. O. Harrison.

WILKINSON, RICHARD H.  
c/o Ecole Nationale Veterinaire, D'Alfort-Seine, France.  
D.V.M., Middlesex University, 1945, and Ecole Nationale Veterinaire, 1956.  
Vouchers: S. A. Vezey and J. N. Beasley.

### Second Listing

BAIN, A. MURRAY, Chivers, Scone, N. S. W., Australia.

KELLER, HANS, c/o Veterinaria A. G., Eibenstrasse 9, Zurich, Switzerland.

KNIGHT, ROBERT P., The Glenormiston Butter & Cheese Factory Co., Ltd., Terang, Victoria, Australia.

MILLER, WALTER W., 2145 Santa Clara Ave., Alameda, Calif.

PESSIN, ARNOLD G., Box 298, Lexington, Ky.

SHEEHY, ROBERT W., Quarters 154 S., Fort Rosecrans, San Diego, Calif.

SUNARA, ANTHONY, 2050 W. LeMoine St., Chicago, Ill.

THOMAS, JULIAN H., 487 Medical Detachment, APO 403, New York, N. Y.

WRIGHT, ALBERT D., 1161 Ransome Dr., Novato, Calif.

Farmers are advised to buy "disease protection from competent veterinarians rather than buying cures"; that is, invest to prevent the fire rather than to put it out.—*Successful Farm.*, Feb., 1957.



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## APPLICATIONS

### Applicants—Members of Constituent Associations

In accordance with paragraph (b) of Section 2, Article X, of the Administrative Bylaws, as revised at the annual meeting of the House of Representatives, Aug. 18, 1951, in Milwaukee, Wis., the names of applicants residing within the jurisdictional limits of the constituent associations shall be published once in the JOURNAL.

The following applicants have been certified as members of the constituent association that has jurisdiction over the area in which the applicant resides. The certification was made by the secretary of the constituent association in accordance with Section 2, Article X, of the Administrative Bylaws.

DOWNIE, HARRY G.  
Department of Research, Ontario Veterinary College, Guelph, Ont.  
D.V.M., Ontario Veterinary College, 1948.

DUMPIS, TALIVALDIS  
Beulah, N. Dak.  
D.V.M., University of Latvia, Riga, 1937.

GLUZ, EMIL  
825½ Spring St., Quincy, Ill.  
D.V.M., Bruna Veterinary College, Czechoslovakia, 1926.

KOVATS, ARTHUR R.  
12609 Buckeye Rd., Cleveland, Ohio.  
D.V.M., University of Budapest and University of Berlin, Germany, 1949.

LINDSAY, SAMUEL G.  
Bonaparte, Iowa.  
D.V.M., Kansas City Veterinary College, 1916.

LITT, ROBERT P.  
8449 S. Chicago Ave., Chicago, Ill.  
D.V.M., Kansas State College, 1946.

PALIONIS, TADAS  
7135 W. Bottsford Ave., Milwaukee, Wis.  
D.V.M., Hannover Veterinary College, 1950.

SWEZEY, I. REEVE  
24th & Providence Ave., Chester, Pa.  
V.M.D., University of Pennsylvania, 1945.

SHIPMAN, NEIL T.  
3816 E. State St., Sharon, Pa.  
D.V.M., Ohio State University, 1947.

VALIUSKIS, ANTANAS  
1832 Greenwood Dr., Ottuma, Iowa.  
D.V.M., Hannover Veterinary College, Germany, 1950.

VINKEL-JENSEN, HANS P.  
9162 N. Swan Circle, Brentwood, Mo.  
D.V.M., Royal Danish Agricultural College, 1937.

WELLMAN, WILLIAM  
116 Grand, Allegan, Mich.  
D.V.M., Michigan State University, 1956.

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## COMING MEETINGS

Central Canada Veterinary Association. Annual meeting. Brockville, Ont., March 15-16, 1957. C. K. Hetherington, 631 Edison Ave., Ottawa, Ont., secretary.

Alabama Veterinary Medical Association. Annual meeting. Lyons Hotel, Decatur, March 17-19, 1957. M. K. Heath, Alabama Polytechnic Institute, Auburn, secretary.

Washington, State College of. Annual conference for veterinarians. Pullman, Wash., April 8-10, 1957. Ray E. Watts, conference secretary.

American Animal Hospital Association. Annual meeting. Sheridan Palace Hotel, San Francisco, Calif., May 8-11, 1957. W. H. Riser, 5335 Touhy Ave., Skokie, Ill., secretary.

Kansas State College. Conference for veterinarians. School of Veterinary Medicine, Manhattan, May 26-28, 1957. E. E. Leasure, dean.

Texas A. & M. College. Conference for veterinarians. Texas A. & M. College, College Station, June 6-7, 1957. R. D. Turk, chairman.

North Dakota Veterinary Medical Association. Annual meeting. Minot, N. Dak., June 17-18, 1957. Dean Flagg, 202 Teton Ave., Bismarck, N. Dak., secretary.

Canadian Veterinary Medical Association. Annual meeting. Vancouver, B.C., July 22-23, 1957. Claude Kealey, 1195 Wellington St., Ottawa 3, Ont., secretary.

Kentucky Veterinary Medical Association. Annual meeting. Brown Hotel, Louisville, July 15-16, 1957. Robert H. Singer, 136 Shawnee Place, Lexington, Ky.

American Veterinary Medical Association. Annual meeting. Cleveland Auditorium, Cleveland, Ohio, Aug. 19-22, 1957. J. G. Hardenbergh, 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

New England Veterinary Medical Association. Annual meeting. Equinox House, Manchester, Vt., Oct. 6-9, 1957. C. Lawrence Blakely, 180 Longwood Ave., Boston, Mass., secretary.

Southern Veterinary Medical Association. Annual meeting. Hotel Roanoke, Roanoke, Va., Oct. 27-30, 1957. A. A. Husman, P. O. Box 91, Raleigh, N. Car., secretary.

A Shetland pony more than 38 years old is owned by a boy in Indiana.—*Prairie Farmer, Jan. 19, 1957.*

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## Regularly Scheduled Meetings

**ALABAMA**—Central Alabama Veterinary Association, the first Thursday of each month. B. M. Lauderdale, Montgomery, secretary.

Jefferson County Veterinary Medical Association, the second Thursday of each month. S. A. Price, 213 N. 15th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the first Tuesday of each month. W. David Gross, 771 Holcombe Ave., Mobile, Ala., secretary.

**ARIZONA**—Central Arizona Veterinary Medical Association, the second Tuesday of each month. Keith T. Maddy, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. E. T. Anderson, Rt. 2, Box 697, Tucson, Ariz., secretary.

**CALIFORNIA**—Alameda Contra Costa Veterinary Medical Association, last Wednesday of each month. Leo Goldston, 3793 Broadway, Oakland 11, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of each month. E. Paul, Redwood City, Calif., secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of each month. A. L. Irwin, 301 Taft Highway, Bakersfield, Calif., secretary.

Mid-Coast Veterinary Medical Association, the first Thursday of every even month. W. H. Rockey, P. O. Box 121, San Luis Obispo, Calif., secretary.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. Lewis J. Campbell, 90 Corral de Tierra, Salinas, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Co-

vell, in Modesto, Calif. Lyle A. Baker, Turlock, Calif., secretary.

Orange Belt Veterinary Medical Association, the second Monday of each month. Chester A. Maeda, 766 E. Highland Ave., San Bernardino, Calif., secretary.

Orange County Veterinary Medical Association, the third Thursday of each month. Donald E. Lind, 2643 N. Main St., Santa Ana, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of each month. T. D. Harris, San Mateo, Calif., secretary.

Redwood Empire Veterinary Medical Association, the third Thursday of each month. Robert E. Clark, Napa, Calif., secretary.

Sacramento Valley Veterinary Medical Association, the second Wednesday of each month. W. E. Steinmetz, 4227 Freeport Blvd., Sacramento, Calif., secretary.

San Diego County Veterinary Medical Association, the fourth Tuesday of each month. H. R. Rossoll, 1795 Moore St., San Diego, Calif., secretary.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, secretary.

Santa Clara Valley Veterinary Association, the fourth Tuesday of each month. Kay Beulley, N. Fourth and Gish Rd., San Jose, Calif., secretary.

Southern California Veterinary Medical Association, the last Wednesday of each month. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

Tulare County Veterinarians, the second Thursday of each month. R. B. Barsaleau, 2333 E. Mineral King, Visalia, Calif., secretary.

**COLORADO**—Denver Area Veterinary Society, the fourth Tuesday of every month. Richard C. Tolley, 5060 S. Broadway St., Englewood, Colo., secretary.



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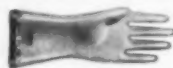


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Northern Colorado Veterinary Medical Society, the first Monday of each month. M. A. Hammariund, School of Veterinary Medicine, Colorado A. & M. College, Fort Collins, Colo., secretary.

**DELAWARE**—New Castle County Veterinary Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. E. J. Hathaway, Clifton Park Manor, Apt. 73-5, Wilmington 2, Del., secretary.

**FLORIDA**—Central Florida Veterinary Medical Association, the second Friday of each month, time and place specified monthly. James B. Murphy, Eustis, Fla., secretary.

Jacksonville Veterinary Medical Association, the second Thursday of each month, time and place specified monthly. George F. Yopp, 4644 Main St., Jacksonville, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. Harold A. Tennant, Atmore, Ala., secretary.

Palm Beach Veterinary Society, the last Thursday of each month in the county office building at 810 Datura St., West Palm Beach. Ross E. Evans, 5215 S. Dixie Highway, West Palm Beach, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. Paul J. Myers, Winter Haven, Fla., secretary.

South Florida Veterinary Society, the third Tuesday of each month, at the Seven Seas Restaurant, Miami, Fla. E. D. Stoddard, 6432 S. W. 8th St., Miami, Fla., secretary.

Suwannee Valley Veterinary Association, the third Friday of each month, at the Thomas Hotel, Gainesville, Fla. R. C. Mann, Rt. 1, Box 37, Ocala, Fla., secretary.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. A. E. Hixon, 131 Mary St., Daytona Beach, Fla., secretary.

**GEORGIA**—Atlanta Veterinary Society, the second Tuesday of every month at the Elks Home on Peachtree St., Atlanta. Ga. J. L. Christopher, Smyrna, Ga., secretary.

**ILLINOIS**—Chicago Veterinary Medical Association, the second Tuesday of each month. Mark E. Davenport, Jr., 215 S. Edgewood Ave., LaGrange, Ill., secretary.

Eastern Illinois Veterinary Medical Association, the first Thursday of March, June, September, and December. A one-day clinic is held in May. H. S. Bryan, College of Veterinary Medicine, University of Illinois, Urbana, secretary.

**INDIANA**—Central Indiana Veterinary Medical Association, the second Wednesday of each month. Peter Johnson, Jr., 4410 N. Keystone Ave., Indianapolis 5, secretary. Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. J. M. Carter, 3421 S. Main St., Elkhart, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P. O. Box 52, Pendleton, Ind., secretary.

**IOWA**—Cedar Valley Veterinary Association, the second Monday of each month, except January, July, August, and October, at Black's Tea Room, Waterloo, Iowa. H. V. Henderson, Reinbeck, Iowa, secretary.

Coon Valley Veterinary Association, the second Wednesday of each month, September through May, at the Bradford Hotel, Storm Lake, Iowa. D. I. Lee, Sac City, Iowa, secretary.

Fayette County Veterinary Association, the third Tuesday of each month, except in July and August, at Pa and Ma's Restaurant, West Union, Iowa. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

**KENTUCKY**—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. L. S. Shirrell, Versailles Rd., Frankfort, secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday evening of each month in Louisville.



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*in ketosis*

## *the way it was*

"This continues to be an important problem to the veterinarian in dairy cattle practice. Some years ago, it appeared to be primarily a disturbance of such a nature that the body was unable to metabolize the fats—more carbohydrate was needed, more ready energy. Many cases responded to glucose intravenously, supported by the feeding of cane molasses or corn syrup. Others responded when chloral hydrate was administered, its effects being to liberate the glycogen stored in the liver.

"There were, however, cases which did not respond to treatment of this nature, but did improve markedly when anterior

pituitary extract was injected. This still did not produce the desired result in all cases, and massive doses of vitamin A were administered. Results were spectacularly good in some cases, but dismally poor in others. When treatment can be accompanied by a change to new pasture, remarkable recoveries may be seen, but obstinate cases still occur in animals that are pasturing on lush green legumes.

"So the search for the underlying cause, and for a means of preventing or correcting the condition, still goes on."

Report of the Committee on Diseases of Dairy Cattle, R. C. Klusendorf, Chairman, J.A.V.M.A. 107:355 (Nov.) 1945.

1945

## *the way it is*

"There appears to be general agreement among those who have studied the disease (primary ketosis) that prompt stimulation of glycogenesis or glucose therapy constitutes the most effective treatment...The satisfactory response of a high percentage of cows treated with 100 mg. prednisolone indicates

that this is adequate dosage. Less severely affected cows may require only 50 mg. It is, therefore, effective in lower dosage than other presently available glucocorticoids recommended for treatment of ketosis."

Lick, R. F.; Norton, D. L., and Huber, W. G. Paper presented at 52nd Annual Meeting, A.V.M.A., Oct. 15-18, 1956, San Antonio, Texas.

1957

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Available in vials of 10 cc.; 10 mg. of prednisolone per cc.

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**MARYLAND**—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m. at the Park Plaza Hotel, Charles and Madison Sts., Baltimore, Md. Harry L. Schultz, Jr., 9011 Harford Rd., Baltimore, Md., secretary.

**MICHIGAN**—Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert E. Kader, 5034 Armstrong Rd., Lansing 17, Mich., secretary.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. S. Correll, Rt. 1, Midland, Mich., secretary.

Southeastern Veterinary Medical Association, the fourth Wednesday of every month, September through May. Gilbert Meyer, 14003 E. Seven Mile Rd., Detroit 5, Mich., secretary.

**MISSOURI**—Greater St. Louis Veterinary Medical Association, the first Friday of the month (except July and August) at the Sheraton Hotel, Spring Ave. and Lindell Blvd. Allen B. Shopmaker, 136 N. Meramec, Clayton 5, Mo., secretary.

Kansas City Small Animal Hospital Association, the first Monday of each month, at alternating hospitals. W. F. Noland, 7504 Metcalf, Overland Park, Kan., secretary.

Kansas City Veterinary Medical Association, the third Tuesday of each month at Exchange Hall, ninth floor, Livestock Exchange Bldg., 1600 Genessee St., Kansas City, Mo. Bush Meredith, 800 Woodswether Rd., Kansas City 5, Mo., secretary.

**NEW JERSEY**—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J. David C. Tudor, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April at the Academy of Medicine, 91 Lincoln Park South, Newark, N. J. Myron S. Arlein, 2172 Milburn Ave., Maplewood, N. J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Casa Mana in Teaneck. James R. Tanzola, Upper Saddle River, secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. F. B. Duke, 49 Taylor St., High Bridge, N. J., secretary.

Southern New Jersey Veterinary Medical Association, the third Tuesday of each month at the Collingswood Veterinary Hospital, Collingswood. W. E. Snyder, E. Kings Highway and Munn Ave., Haddonfield, secretary.

**NEW YORK**—New York City, Inc., Veterinary Medical Association of the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St., New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

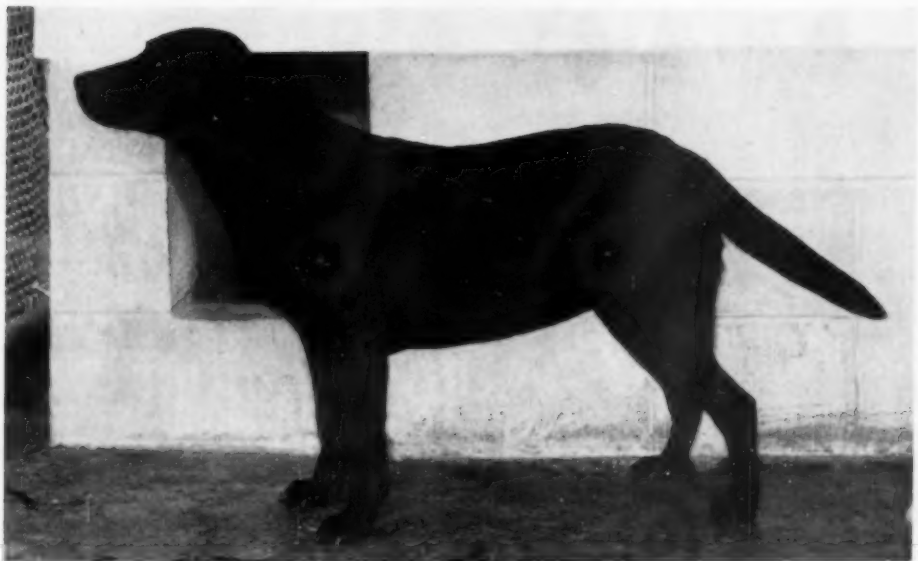
Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

**NORTH CAROLINA**—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Heary Hotel, Greensboro. Joseph A. Lombardo, 411 Woodlawn Ave., Greensboro, secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month. Wm. Allen Potts, 401 W. James St., Mount Olive, secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. John G. Martin, Boone, N. Car., secretary.

**OHIO**—Cuyahoga County Veterinary Medical Association, the first Wednesday of each month, September through May (except January), at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. Ed. R. Jacobs, 5322 Pearl Rd., Cleveland, Ohio, secretary.



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Many dog experts feel that dry dog food should be supplemented by fresh meat. And the makers of Friskies recognize that the addition of meat is a widely accepted practice among dog breeders, kennel men and dog owners.

**HOWEVER**, in the case of Friskies, we've found in test after test that adding fresh meat to Friskies Meal is not necessary. Friskies in its meal form, actually supplies all the nourishment of finest fresh meat—plus the other necessary food elements, vitamins and minerals for a complete and fully balanced dog diet.

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**OKLAHOMA**—Oklahoma County Veterinary Medical Association, the second Wednesday of every month. James M. Brown, 2818 W. Britton Rd., Oklahoma City, secretary.

**Tulsa** Veterinary Medical Association, the third Thursday of each month in Directors' Parlor of the Brookside State Bank, Tulsa, Okla. Don L. Hohmann, 538 S. Madison St., Tulsa, Okla., secretary.

**PENNSYLVANIA**—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine, 39th and Woodland Ave., Philadelphia 4, Pa. Raymond C. Snyder, 39th and Woodland Ave., Philadelphia 4, Pa., secretary.

**SOUTH CAROLINA**—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

**TEXAS**—Coastal Bend Veterinary Association, the second Wednesday of each month. J. Marvin Prewitt, 4141 Lexington Blvd., Corpus Christi, Texas, secretary.

**VIRGINIA**—Central Virginia Veterinarians' Association, the third Thursday of each month at the William Byrd Hotel in Richmond at 8:00 p.m. M. R. Levy, 312 W. Cary St., Richmond 20, Va., secretary.

**Northern Virginia Veterinary Society**, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

**Southwest Virginia Veterinary Medical Association**, the first Thursday of each month. I. D. Wilson, Blacksburg, secretary.

**WASHINGTON**—Seattle Veterinary Medical Association, the third Tuesday of each month in the Trinity Episcopal Church, 8th and James St., Seattle, Wash. P. R. Des Rosiers, 5508 2nd Ave., N. W., Seattle 7, secretary.

**South Puget Sound Veterinary Association**, the second Thursday of each month except July and August. O. L. Bailey, P. O. Box 906, Olympia, Wash., secretary.

**WEST VIRGINIA**—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the second Thursday of each month in the Hotel Prichard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

**WISCONSIN**—Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. George F. Lynch, 201 West Devon St., Milwaukee 17, Wis., secretary.

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of small animals



FURADANTIN is one of the nitrofurans—a unique class of antimicrobials, unrelated to antibiotics or sulfonamides. Like all the nitrofurans, FURADANTIN is bactericidal to a wide range of both gram-negative and gram-positive organisms. It is nontoxic to kidneys, liver and blood-forming organs. Development of bacterial resistance to FURADANTIN is negligible.

In canine tracheobronchitis (kennel cough), FURADANTIN stopped the cough in 11 of 12 dogs within 3 days. Within 1 week, all were fully recovered. There were no recurrences.<sup>1</sup>

1. Masler, J. E.: Vet. M. 30:803, 1935. 2. Breaker, E. S.; Holt, S. H., and Siegel, D.: J. Michigan M. Soc. 54:803, 1935.

**Dose:** 1 to 2 mg. per lb. of body weight, 3 times daily, for 4 to 7 days.


In urinary tract infections of dogs and cats, FURADANTIN rapidly produces high antibacterial concentrations in urine. As reported on urinary tract infections in humans, "it appears that Furadantin is one of the most effective single agents available at this time."<sup>2</sup>

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action at  
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*acetate veterinary  
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Suspension, brand of prednisolone acetate,  
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1 dose-5 x 1 dose-10 doses-100 doses  
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1 dose (4 cc.)  
*Other products to be added.*

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Young, ambitious veterinarian seeks assistantship leading to possible partnership; or position with firm, school, or state requiring diagnostic and/or field service work. Address "Box E 14," c/o JOURNAL of the AVMA.

Cornell graduate with mixed practice for 5 years desires position in small animal hospital in eastern state. Married, 3 children. Address "Box E 15," c/o JOURNAL of the AVMA.

Graduate, 1954, desires position in small animal practice; 1 year small animal experience. Licensed in

Virginia, North Carolina, Florida; available July. Address "Box E 17," c/o JOURNAL of the AVMA.

#### For Sale or Lease—Practices

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Lucrative small animal practice for sale in fast-growing New England town. Established 20 years; excellent clientele. Comfortable 7-room house; neat kennel buildings; 50 cages; 6 1/2 acres, landscaped; drugs and equipment. Retiring. Address "Box E 6," c/o JOURNAL of the AVMA.

Small animal hospital for sale; well-established, excellent location, Piedmont, N.C.; 35 kennels and outside runs. Gross, \$20,000; price, \$13,500, includes hospital, large lot, drugs, and equipment. Will finance \$7,500. Address "Box E 11," c/o JOURNAL of the AVMA.

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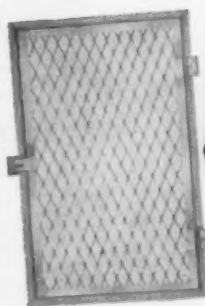
Practice for sale, county seat town, southeastern Iowa; prosperous agricultural area, excellent town in which to live. Practice 50% large animals—swine, beef, and dairy; 50% small animals. Hospital contains 26 kennels and outside runs. Gross, \$25,000; owner will finance. Address "Box E 16," c/o JOURNAL of the AVMA.

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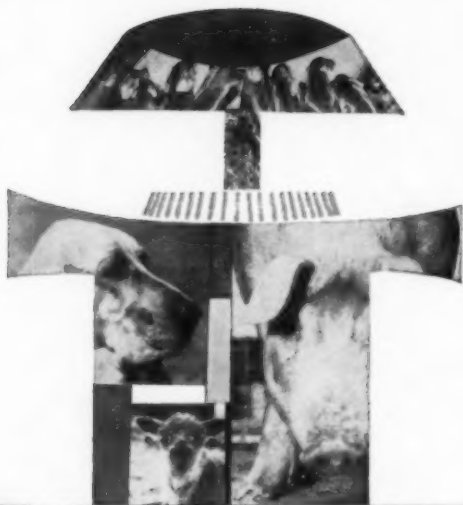
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